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POIKILODERMATOMYOSITIS

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Under the title "Sclerose atrophique de la peau et myosite généralisée," Petges and Clégat [1] in 1906 described a case of myositis with cutaneous atrophy and poikiloderma similar to that described, about the same time, by Jacobi.[2] In 1930 G. Petges and A. Petges [3] reviewed the subject and reported 2 additional cases under the title "Poikilodermatomyosite."

Dermatomyositis, so named by Unverricht,[4] follows a course similar to the foregoing condition but lacks the poikiloderma. Numerous reports of dermatomyositis appear in the literature, and poikilodermatomyositis is discussed at length on several occasions, but actual case reports are rare. The parallelism of the condition in the case to be reported to dermatomyositis, except with reference to the superficial cutaneous changes, leads us to the belief that poikiloderma results from an unusually severe process and that dermatomyositis and poikilodermatomyositis are variants of the same process.

REPORT OF A CASE

Mrs. F., aged 42, was admitted to the Allegheny General Hospital on Oct. 29, 1937, complaining of severe asthenia, muscular soreness and an eruption. She had been in good health until five years before, at which time, after influenza,

From the Allegheny General Hospital and the William H. Singer Memorial Research Laboratory.

Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 1, 1939.

1. Petges, G., and Clégat, C.: Sclerose atrophique de la peau et myosite généralisée, Ann. de dermat. et syph. **7**:560, 1906.

2. Jacobi, E.: Fall zur Diagnose (Poikilodermia vascularis atrophicans), Verhandl. d. deutsch. dermat. Gesellsch. **9**:321, 1907.

3. Petges, G., and Petges, A.: Poïkilodermatomyosite dans la jeunesse et l'enfance, Ann. de dermat. et syph. **1**:441, 1930.

4. Unverricht: Dermatomyositis acuta, Deutsche med. Wchnschr. **17**:41, 1891.

she experienced increasing weakness and had frequent attacks of an infection of the upper respiratory tract, which recurred at the time of the onset of her present condition. This had been present for ten months before admission to the hospital. In January 1937 she noticed a slight redness on the right cheek, and on the following day the area about the left eye was edematous and red. The swelling and redness gradually became worse and involved the lids and surrounding areas of both eyes. At the same time she noticed soreness about the elbows, which seemed to be more in the ligaments and muscles than in the joints. The skin about the axillas became inflamed and edematous. Successive muscle groups became involved, and there was gradual extension of the dermatitis.

At the time of admission the face, neck, scalp, arms and hands and the back as far as the inferior angles of the scapulas were involved. She had noted gradual thinning of the hair of the scalp and eyebrows, a generalized feeling of pain, stiffness and chilly sensations but no chills. She was so weak she could not turn in bed unassisted. Her temperature, which on admission was 99 F., usually ranged from 99 to 101 but at intervals reached 105 following chills. These exacerbations were accompanied by increased severity of the cutaneous and general symptoms. Two weeks after admission purulent bronchitis developed, which, however, did not become bronchopneumonia. At irregular intervals paroxysmal tachycardia was present, but the electrocardiograms were normal. Her general condition became gradually worse until April, at which time death seemed imminent on several occasions. About the middle of the month improvement began, and early in May her temperature reached normal and remained so. She was discharged from the hospital May 25, at which time her skin generally presented alternating areas of atrophy, ectasia and pigmentation. There was some contracture at the knees and elbows, which gradually responded to passive motion, massage and extension. She gradually gained in weight and strength and during June began to walk about the house.

A striking feature of this case was the progress of the cutaneous condition while the patient was in the hospital. Approximately palm-sized areas of her skin would become acutely inflamed, producing a dermatitis which was erythematous and was soon covered by fine, dry scales. In about two weeks ectatic vessels would appear, and in about two months the dermatitis would subside leaving the skin atrophic and with considerable ectasia and pigmentation simulating roentgen ray burns. Symmetric and asymmetric new areas of dermatitis appeared at varying intervals, and at times these were accompanied by exacerbation of the underlying myositis. In each instance there were an elevation of the patient's temperature and an aggravation of her general condition. Single bullae appeared early in the cycle on three occasions.

Laboratory Data.—Shortly after the patient's admission a blood count revealed 394,800 platelets, 4,550,000 red corpuscles, 11,000 white corpuscles, 75 per cent hemoglobin, 55 per cent polymorphonuclears, 19 per cent stab cells, 18 per cent lymphocytes, 5 per cent monocytes, 1 per cent basophils and 2 per cent eosinophils. Subsequent blood counts failed to show any decided change in any of the elements save in the eosinophils, the percentage of which remained between 1 and 2 until five months after admission, when it rose to 19 per cent and subsequently to 28 per cent. At the same time the total count of white cells was only 7,400.

The blood chemistry was within the normal range. The serum sodium and the blood chlorides were unaltered. The blood cholesterol and cholesterol esters were unchanged. Dextrose tolerance tests revealed a greatly decreased tolerance with a high curve. At the end of two hours the blood sugar was 0.20 per cent. The Wassermann and Kahn reactions were negative.

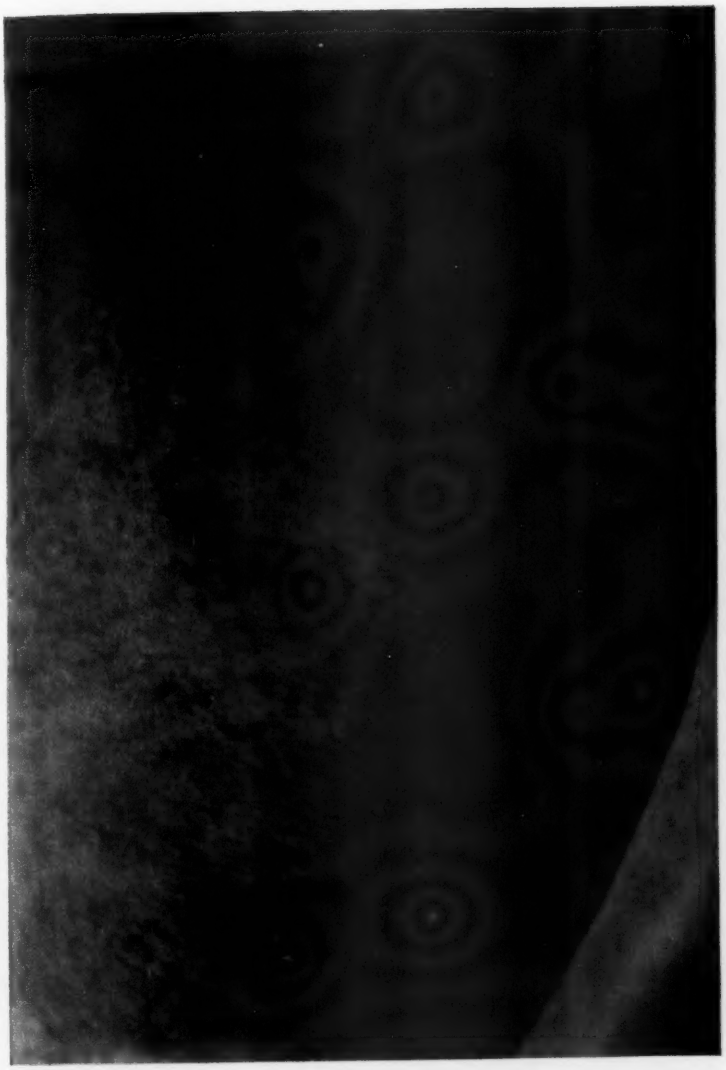

Fig. 1.—Ectasia of the skin and manner of distribution of the lesions.

UNIV. OF MICH.

The basal metabolic rate was + 28 per cent. At the time the test was made the patient's temperature was 99.4 F. and her pulse rate 112.

Blood cultures at five different intervals in six weeks revealed no growth. However, seven months after admission during one of the periods of sudden chills and rising temperature, a blood culture yielded 35 colonies of Streptococcus viridans per cubic centimeter.

Several abscessed teeth were removed, and cultures were made. These yielded Str. viridans and Staphylococcus aureus.

Subsequent cutaneous tests with an autogenous vaccine made from the Str. viridans gave a definitely positive skin reaction. Other bacterial skin tests were all negative.

About the same time the bacterial growth was found in the blood culture, a portion of muscle taken for biopsy was cultured both aerobically and anaerobically, and no growth was obtained, even after thirty days' incubation.

Routine urinalyses gave essentially negative results. At the height of the acute stage of the disease a twenty-four hour specimen of urine showed the presence of 451 mg. of creatine and 499 mg. of preformed creatinine. The creatinuria was indicative of the severe degree of muscle destruction.

Determinations of vitamin C in the blood showed the presence of 0.4 mg. per hundred cubic centimeters three months after the patient's admission to the hospital. As a result of this greatly diminished content, 100 mg. of ascorbic acid was administered intravenously three times daily, and copious amounts of orange juice were given by mouth. After six weeks the ascorbic acid content in the blood rose to 1.4 mg. per hundred cubic centimeters. The administration of ascorbic acid was stopped completely, and within ten days the vitamin C content of the plasma dropped to 0.70 mg. The vitamin therapy was then resumed, and the vitamin C content of the plasma rose promptly to 1.7 mg. per hundred cubic centimeters.

The first biopsy was performed shortly after the patient's admission to the hospital. A portion of skin that showed a papulosquamous eruption of bluish gray was examined. Microscopic examination revealed the surface covered by a considerable amount of keratinized epithelium. The rete pegs were flattened. One area of the epidermis showed early atrophy. There was a moderate degree of collagenous hyalinization. The small arterioles showed some ectasia and perivascular infiltration with lymphocytes and occasional eosinophils. The same type of inflammatory reaction was present about the ducts of the sebaceous and sudoriparous glands.

A second specimen of skin for biopsy was taken several months later from the region of the breast. Histologic examination showed the changes that had occurred in the skin with the passing of time. The epidermis showed a more advanced degree of keratinization and atrophy. There was much further advanced hyaline degeneration of the collagen bundles in the corium than was seen in the specimen taken several months before. No inflammatory cells were observed, and the whole specimen represented a portion of dense, hyalinized collagenous material which was covered by an atrophic, keratinized, flattened epidermis. Gram-Weigert stains were made of both sections of skin, but no organisms could be demonstrated.

A section of muscle from the calf of the leg was removed at the time the patient had a Str. viridans septicemia. This was examined, and sections were made and stained with phloxin and methylene blue, with hematoxylin and eosin, with Mallory's hematoxylin–phosphotungstic acid stain and with the Gram-Weigert stain for organisms. Microscopic examination showed considerable edema and

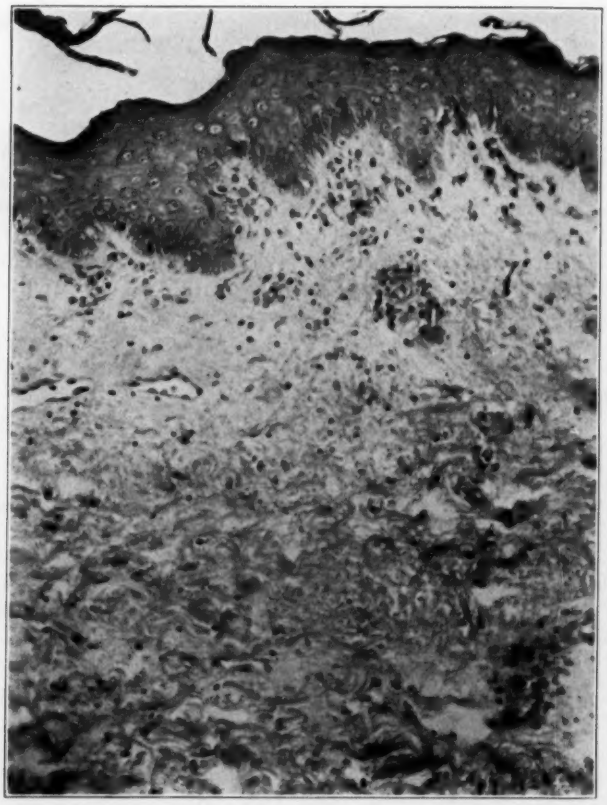

Fig. 2.—Early lesion of the skin revealing beginning ectasia in the cutis.

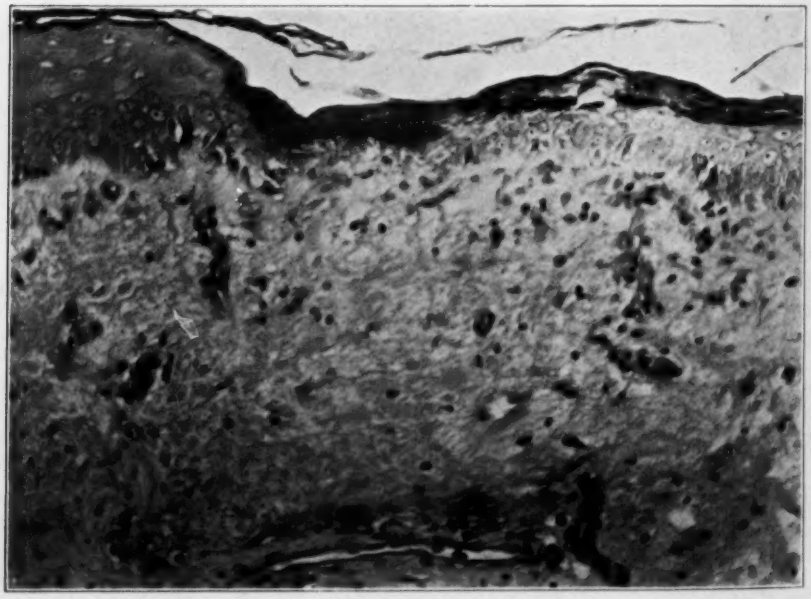

Fig. 3.—Advancing lesion of the skin showing keratinization and atrophy of the epidermis and ectasia of the deeper vessels.

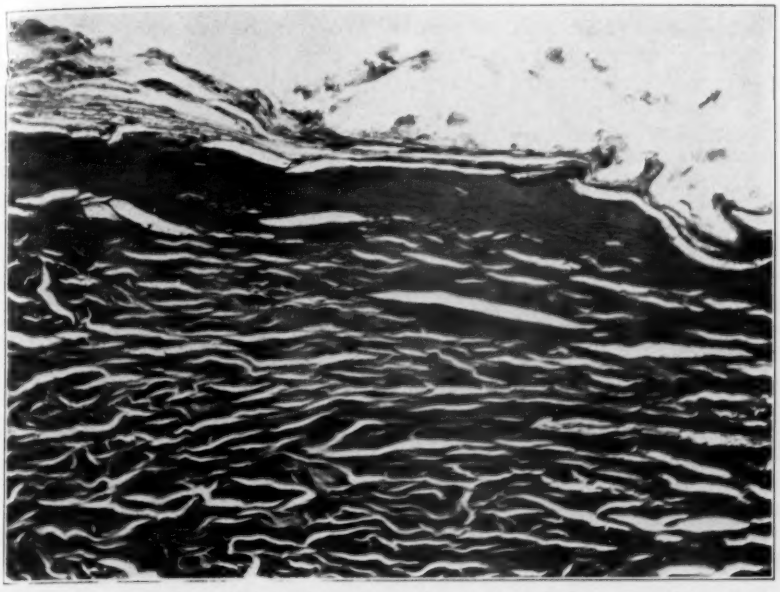

Fig. 4.—The end result in a far advanced cutaneous lesion. Complete atrophy of the epidermis and hyalinized collagen in the cutis.

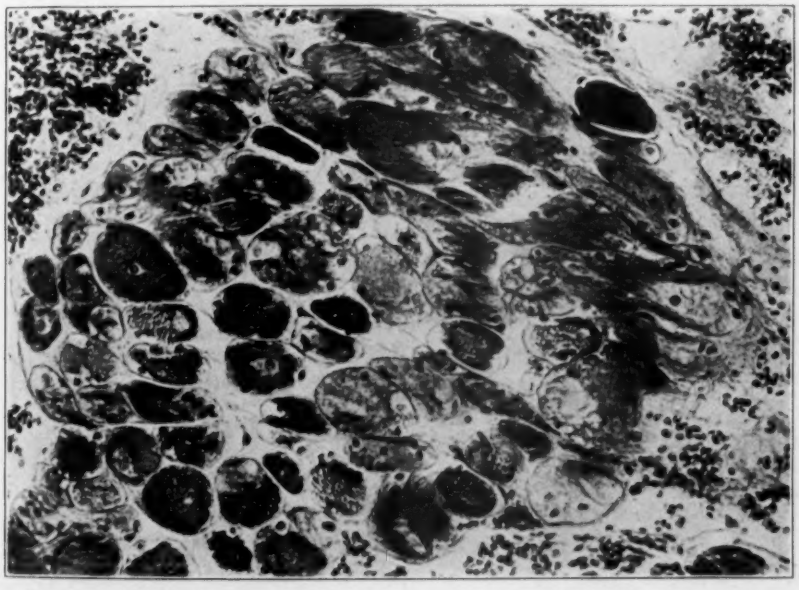

Fig. 5.—Section of muscle showing intercellular, hemorrhagic infiltration and edema of the perimysium and endomysium. Note perinuclear vacuolization.

hemorrhagic infiltration of the perimysium which extended into the endomysium, so that the muscle fibers stood out prominently against the hemorrhagic background. There were varying degrees of degenerative changes in the muscle fibers. These varied from simple, cloudy swelling to an advanced degree of vacuolation. The vacuoles were most prominent in the perinuclear portion and caused the sarcolemmas to be raised into irregularly outlined blebs. The muscle fibers stained unevenly and in many places showed a total loss of cross striations. A mild degree of fibrous connective tissue replacement of the muscle fibers was observed, but this had not yet progressed very far in the section which was available for examination.

One of the most striking changes was the compact perivascular lymphocytic infiltration of the smaller arteries. This was similar to that seen in cases of periarteritis nodosa. In the areas of diffuse hemorrhagic involvement, there was also slight interstitial eosinophilic infiltration.

COMMENT

There are several factors which strengthen the consideration of poikilodermatomyositis as a variant of the simpler dermatomyositis. By postulating the pathogenesis of dermatomyositis, one can arrive at a better understanding of the association of the poikiloderma with the myositis and cutaneous disturbances which together give rise to the symptom complex with which we are dealing. Reference to the microscopic pictures of the biopsy specimens of skin and muscle shows the chief lesion to be a vascular one involving the superficial cutaneous and subcutaneous vessels as well as the vessels within the muscles. In the early lesions there is ectasia in the vessels of the cutis, associated with a hemorrhagic extravasation. The same process, occurring through diapedesis, produces a similar condition within the muscles. The severity of the process probably causes an extravasation of blood through an increased vascular permeability. When this occurs in the superficial vessels, it manifests itself as an ectasia with poikiloderma. When it occurs in the vessels of the muscle, one sees changes similar to those observed in cases of generalized myositis ossificans, in which the aftermath consists of an acute and chronic inflammatory cellular exudate plus fibrous replacement of the degenerated muscle.

When the process is not acute but goes on to chronicity, as in our case, there is atrophy of the epidermis and cutis, with the development of hyalinized collagen and consequent compression of the blood vessels. As a result, areas of atrophy of the skin with pigmentation are observed. Thus, the severity of the process plus a probable altered metabolic state has brought about a condition of poikiloderma in dermatomyositis.

These thoughts lead to the consideration of the various factors involved in the production of the train of events leading to this symptom complex. Our studies showed the patient to have a low ascorbic acid content of the blood. As a consequence of this, an increased vascular

permeability probably resulted, with hemorrhagic extravasations and the aforementioned sequelae. That a metabolic factor, as yet unexplained in cases of human beings, can be an important consideration was shown by the experimental work of Goettsch and Pappenheimer,[5] who produced similar muscular lesions in rabbits and guinea pigs with special diets. The fact that the administration of large doses of ascorbic acid caused the ascorbic acid content in the blood to return to normal but failed to maintain this state when administration was stopped speaks for disturbed metabolic mechanism. We are fully aware that the low ascorbic acid content in the blood is not sufficient in itself to explain all the changes that occurred, especially since we found actual destruction of muscle, as evidenced by the decided increase of creatine in the urine and by the altered histologic picture of the section of muscle. The report of Wolf and Wilens[6] and their quotations are in agreement with our findings of diminished creatinine and increased creatine content of the urine. They compared the values for creatine and total nitrogen content of muscle cited by Bodansky with those studied in their reported cases.

Though isolated facts appear which tend to throw light on the possible modus operandi of this condition, the underlying etiologic factor remains obscure. Various organisms have been suggested, but no specific one has thus far proved acceptable. In our case, the presence of Str. viridans in the apexes of some of the teeth and its appearance on one occasion in the blood stream, together with the positive reaction to intracutaneous injections of an autogenous vaccine, strongly suggest that the green streptococcus in this particular case was at least a contributing etiologic factor. It is difficult for us to explain the entrance of Str. viridans into the blood stream at a date so long after the extraction of the teeth and in the absence of any other demonstrable focus. It is noteworthy that the extraction of the teeth was followed in each instance by an acute exacerbation of all her symptoms. Cultures of muscle failed to yield a growth.

It is important to consider a few points in the differential diagnosis of this condition. Trichinosis is a condition that must be ruled out, especially when edema of the face and eosinophilia are present, as they were in this case. The cutaneous lesions and the biopsy specimen of muscle are sufficient to eliminate Trichina infestation as a diagnosis. Periarteritis nodosa must also be regarded as a possible diagnosis when a section of muscle shows the perivascular cellular infiltration such as was observed. Many cases of periarteritis nodosa show various bizarre manifestations in the skin which make the differential diagnosis increas-

5. Goettsch, M., and Pappenheimer, A. M.: Nutritional Muscular Dystrophy in the Guinea Pig and Rabbit, J. Exper. Med. **54**:145, 1931.

6. Wolf, A., and Wilens, S. L.: Dermatomyositis: A Report of Two Cases with Complete Autopsy, Am. J. Path. **12**:235, 1936.

ingly difficult. However, the decided creatinuria which is present in dermatomyositis is not found in periarteritis nodosa. Other rare condi-

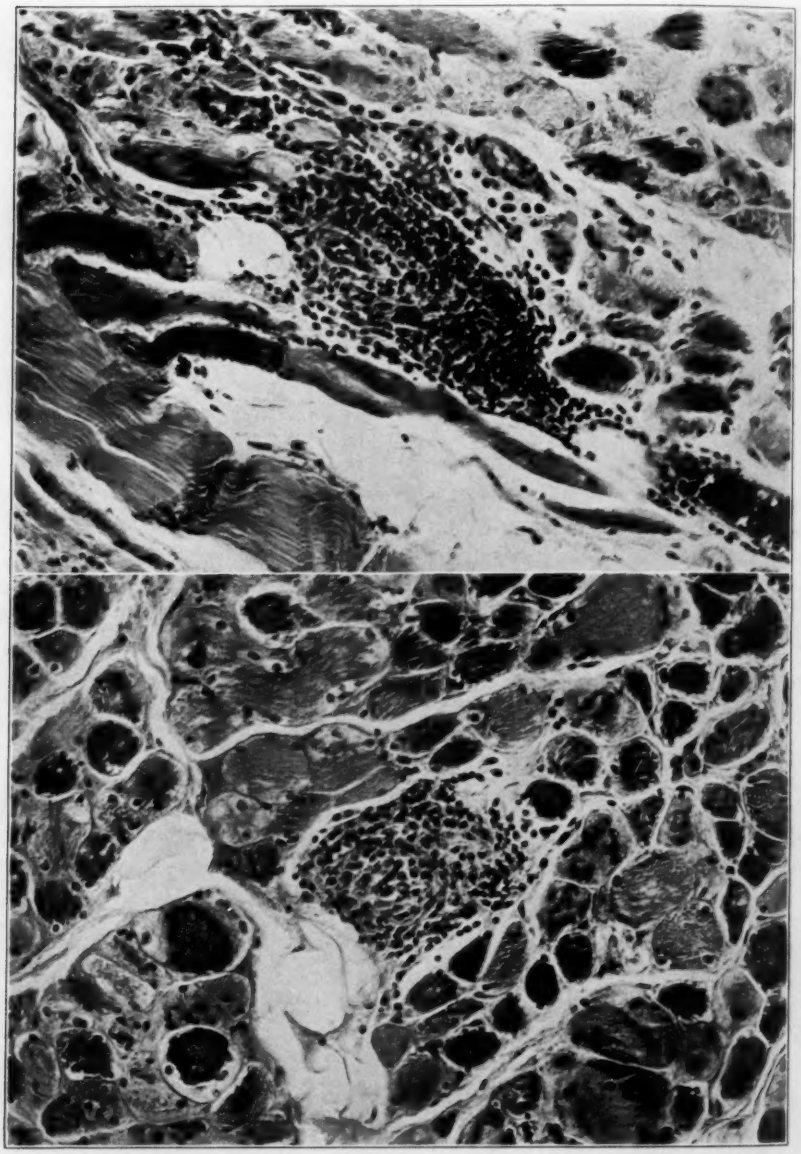

Fig. 6.—Perivascular collars of lymphocytes deep in the muscle bundles. Early cloudy swelling of muscle bundles.

tions in which there is muscular involvement appear to us to be so far removed from a differential diagnostic consideration as not to warrant any discussion.

SUMMARY

A case of poikilodermatomyositis is reported in which there were all the findings of dermatomyositis plus poikiloderma, implying that the latter is a variant of the former.

The histologic pictures explain the clinical manifestations in various stages.

The isolation of Str. viridans from the teeth and blood stream implies a possible etiologic relation in our case.

The depressed ascorbic acid content of the blood suggests the use of vitamin C as a therapeutic agent.

Foci of infection should be removed with extreme caution, owing to the great surgical risk.

ABSTRACT OF DISCUSSION

DR. PAUL A. O'LEARY, Rochester, Minn.: About ten years ago Dr. Otto Foerster first called the attention of the members of this society to the fact that poikiloderma atrophicans vasculare is occasionally associated with mycosis fungoides. Since then I have seen poikiloderma atrophicans vasculare not only in association with mycosis fungoides but in association with disseminated lupus erythematosus and with dermatomyositis. Dermatomyositis has been observed in association with other dermatoses, such as mild degrees of exfoliative dermatitis, and with a disseminated eruption, which is clinically difficult to distinguish from lupus erythematosus disseminatus. I have also seen dermatomyositis in patients with addisonian-like pigmentation in addition to those who do not display any characteristic cutaneous disease. Accordingly, dermatomyositis may be seen in association with a variety of cutaneous pictures for which there is no known common etiologic factor.

I like to look on dermatomyositis as a primary myositis in which the cutaneous disease is not only secondary but varied. In the cases in which my associates and I obtained specimens of muscle, the histologic picture varied from an acute inflammatory process in the early stage to almost complete destruction of the muscle in the far advanced severe types. In a few cases of the early phases of the disease streptococci have been cultured from the muscle. These organisms were found, however, only in cases of acute involvement, and as the disease became older we were unable to obtain positive cultures. An autogenous vaccine given to a few patients seemed at first to have definite merit; however, our enthusiasm cooled when other patients recovered spontaneously. I believe it advisable to bear this in mind when one endeavors to appraise any therapeutic measure for dermatomyositis. I, too, noted the leukopenia and the practically normal sedimentation rate, so that the evidence to support an infectious process, in spite of the fact that in some cases we cultured streptococci from the muscle, is not conclusive. I want to call attention to the inability to describe the typical pathologic picture in the muscles in dermatomyositis. This is due, of course, to the fact that the process in the muscle starts out as an acute inflammatory condition and ends in degeneration of the muscle tissue. I think also that confusion is sometimes caused by the finding of high degrees of eosinophilia in some cases. I have observed an eosinophil count of 35 per cent, and repeated search failed to demonstrate trichinas. I like to look on poikiloderma as a disease which has been produced by the effect

of various toxins on the vascular tree. The effect may be limited to the blood vessels of the skin, as in the cases of milder involvement, or it may be severe enough to include the blood vessels of the viscera.

Dr. C. Guy Lane, Boston: I should like to add a case to this series. The patient was well until 1929, at which time a red, pruritic rash developed on the extensor surface of the right arm and the anterior surface of the neck. This gradually spread, over a period of one and a half years, to involve the entire face, the neck, the lateral aspect of the upper extremities and the dorsa of the hands and fingers. Involvement of the face was associated with swelling of the cheeks and eyelids. In 1932, in the dermatologic department, the condition was diagnosed as a lichen planus-like dermatitis. The description at that time was of involvement as described, the lesions being made up of definite lichenoid papules with typical flat, angular, shiny tops, having a purplish tinge. In 1932 the patient was seen with the complaint of a "swollen leg," diagnosed as phlebitis. In 1933 muscular weakness and loss of weight developed. A report after neurologic consultation at this time stated: "The picture is consistent with progressive muscular atrophy." Swelling of the joints of the fingers, elbows and shoulders also developed, so that a diagnosis of atrophic arthritis was made. There were several admissions to the hospital. In 1935 dermatologic consultation revealed telangiectasis of the nail beds.

The condition suggested disseminated lupus erythematosus, but when combined with the entire cutaneous picture it was more suggestive of poikiloderma atrophicans vasculare. On Feb. 8, 1939, the patient showed an eruption involving the face, neck, upper part of the chest, shoulders, extensor surfaces of the arms and anterior portion of the thighs. The skin in the involved areas was sclerotic and covered by many scattered small blood vessels and pinpoint lesions, which faded under pressure. The eyelids and the periungual area of the fingers showed numerous telangiectatic vessels. All muscles were deformed, and there was limitation of motion at all joints, though the joints themselves did not seem to be affected. Diagnosis at this time was chronic dermatomyositis. The patient has always shown traces of sugar in the urine, an abnormally high blood sugar and massive enlargement of the liver and spleen. Histologic examination revealed dermatomyositis.

Dr. J. Gardner Hopkins, New York: This case belongs to an interesting group. Petges described three syndromes, which he named poikiloderma, poikilodermatomyositis and poikilodermatomyositis with calcification. I recently observed a case which seemed typical of the third group, that of a Negro about 22 years of age. The disease at first sight had little in common with that described by Dr. Guy. It was a chronic process of ten or twelve years' duration with no history of febrile onset. A little itching of the skin was all that could be elicited from the history at first. The patient showed areas of mottled atrophy of the skin with little telangiectasis and distinct markings on the backs of the hands, back of the ears and on the shins, and the muscles felt like bands of old dried out rubber. There were contractures of the hands and arms, so the patient could not straighten them, and he was so weak that he could not walk. In addition, there were calcareous deposits over the joints, and a number of these had worked through the skin. Roentgen examination revealed calcareous deposits in the soft tissues, especially in the axillary folds over the adductors of the thighs and between the metatarsal and the metacarpal bones. The patient also had extreme caries of the teeth and pyorrhea. In the absence of anything else, it was thought the dental condition might bear a causal relation to the cutaneous disease. As in Dr. Guy's

case, extraction of the teeth was followed by high fever and prostration. It is difficult to say whether the condition in such cases represents the chronic form of the disease Dr. Guy has reported or whether the two conditions are distinct.

DR. HARTHER L. KEIM, Detroit: My reason for discussing Dr. Guy's paper is to add another case and also to make a confession. About six months ago a patient presented herself at my office with lichenoid erythroderma, swelling of the eyelids and weakness. I did not classify the condition, thinking that I was dealing with an unusual type of lichen planus. Weakness continued, and the patient was hospitalized. At that time Dr. O'Leary, at one of the local meetings, read a short paper on dermatomyositis. It was then apparent that my patient had the characteristic signs and symptoms of this syndrome. Death ensued eight weeks later, with increasing weakness and edema and finally cardiac failure. A complete postmortem examination was made which showed the characteristic findings together with some unusual degenerative changes in the myocardium. Dr. Jamieson will, I believe, report this case later. I hope the cases discussed here will help bring about the prompt recognition of this condition, as I have the conviction that it is not as rare as the number of reported cases would lead one to believe.

DR. GEORGE M. MACKEE, New York: I should like to mention briefly an unreported case. The patient had an unusual symptom, which is the reason for the report. She was a Jewish woman of 70. Poikiloderma developed, which spread rapidly and became universal in less than six months. At first the patient had chills and fever. When I first saw her these symptoms had subsided, but she had a low grade fever. The white cell count was high. Streptococci were in the throat cultures. The histologic structure was that of poikiloderma. There were no calcareous deposits in the muscles. The muscles were useless because of atrophy and painful myositis. There was profound emaciation. The patient was most unruly and I succeeded in keeping her in the hospital only a couple of weeks. I was able to do little investigative work. The unusual feature was numerous superficial ulcerations. Some were small and superficial, while others were large and deep. The diagnosis in this case was considered to be poikilodermatomyositis.

DR. EDWARD A. OLIVER, Chicago: I do not think that Dr. Keim should feel bad about his failure to diagnose the condition in the case he refers to. When Jacobi presented his first case of poikiloderma, both Jadassohn and Ehrmann made a diagnosis of scleroderma, while Kreibich and Arndt considered the condition lupus erythematosus. There has been just as much confusion in Europe as there has been in this country as to what constitutes the symptoms of poikiloderma atrophicans vasculare. In an interesting paper by Jordan (*Dermat. Wchnschr.* **89**:1829, 1929), the symptoms of a bright red marbling of the skin, telangiectasia, atrophy, especially in localized processes on the head and trunk, adherent scaling and a strong resemblance of the patches to roentgen dermatitis were all emphasized. The fact that Jacobi decleared the condition in his case similar to if not identical with that in the case of Petges and Clijate and that a report of the case was published under the title of sclerose atrophy and generalized myositis gave rise to misconceptions. Jordan, however, believed that in cases of poikiloderma atrophicans vasculare complications such as myositis, scleroderma and even generalized atrophy may occur.

DR. FRED D. WEIDMAN, Philadelphia: The real message of Dr. Guy's presentation has not been discussed, doubtless because a similar experience has not been met. This must explain why the discussion has been confined to a discussion of

individual cases. One should exercise care in determining whether the cutaneous changes are those of true poikilodermatomyositis according to the original conception of that disease. I am prompted to say this because I recently studied sections referred to me by Dr. Oris G. Hazel, who a short time ago read a paper on the subject (Poikiloderma Vasculare Atrophicans: Report of a Case, ARCH. DERMAT. & SYPH. **40**:776 [Nov.] 1939). I was fortunate in being able to see the patient in Oklahoma City, and there was no question about the telangiectasis or about the depressions over the cutaneous surface. Clinically, I mistook them for scars. Dr. Hazel, however, made no less than seven biopsies, and in the sections it was seen that the depressions were not scar tissue formation but simply areas where the infiltration was not the salient feature. In other words, the fundamental disease process was located in the site of the infiltration as viewed clinically, and the intervening places where the infiltration was not present accounted for the confusing scarlike depressions. After I had seen the sections, I concluded that the condition was not poikiloderma but was related somehow to the lymphoblastomas. In substance, then, one should be careful indeed in distinguishing between the true poikilodermic changes and poikiloderma-like ones.

INFLUENCE OF AGE ON RINGWORM INFECTION OF THE SCALP

AN EXPERIMENTAL STUDY

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IN COLLABORATION WITH
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Ringworm of the scalp in children is a commonplace clinical observation. Its low incidence in the years following puberty and in late adult life, at least in Europe and America,[1] is equally well known and to date has no adequate explanation. From time to time various theories have been offered, including the differences in anatomic structure existing between young and adult hair,[2] the virulence and pathogenicity of the invading organism [3] and changes in the hormonal activity of the glands occurring at adolescence.[4] Detailed experimentation following each of these leads and including the various organisms in question would take one far beyond the scope of a single paper. This report therefore deals with but three common organisms chosen arbitrarily. It describes an attempt to demonstrate, by one method only, differences in the chemical contents of young and old hair and their possible influence on the cultural growth of the organisms mentioned.

Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 3, 1939.

From the University of Oregon Medical School (Dr. Kingery) and from the Department of Chemistry, Oregon State Agricultural College (Dr. Williams and Mr. Kidd).

1. Fox, H., and Fowlkes, R. W.: Ringworm of the Scalp in Adults, Arch. Dermat. & Syph. **11**:446 (April) 1925.

2. Thin, G.: Pathology and Treatment of Ringworm, London, J. & A. Churchill, 1887.

3. Sabouraud, R.: Les teignes, Paris, Masson & Cie, 1910; Généralités concernant les dermatophytes: sur le Trichophyton violaceum, Ann. de dermat. et syph. **9**:759, 1928. Bogrow, S. L., and Tschernogubow, N. A.: Trichophyte der Erwachsenen, Dermat. Wchnschr. **57**:1027, 1913.

4. Bloch, B., in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1928, vol. 11, p. 321. Kusunoki, F.: Experimentelle und klinische Studien zur Lehre der Dermatomykosen, Arch. f. Dermat. u. Syph. **114**:1, 1912.

The selection of a suitable medium offered certain difficulties. To those who have concerned themselves to any degree with the specific nutritional requirements of the pathogenic fungi, their vagaries are known only too well. Sabouraud's medium, for example, stands out in its field of general usefulness. Yet an accurate determination of its necessary and unnecessary components almost defies present analytic methods. Thus even a most superficial investigation of the growth requisites of these organisms impresses one with the magnitude of the problem involved. A further fact of considerable moment in studies of this type is the importance of the number of organisms placed in a given medium in determining whether or not growth results. It is an easily demonstrated fact with many mediums that unless a certain minimum number of organisms is introduced no growth results. Observations of this type continued over long periods leave little doubt that in certain instances laboratory reports of "no growth" mean simply insufficient "dosage" in inoculation. This fact should be taken into consideration in every diagnostic procedure of this type. These and similar observations led us to choose a medium which, by experience, we knew contained minimum requirements for growth, for most organisms under ordinary conditions. At the same time the medium had to be such that the addition even of infinitesimal amounts of any chemical, either stimulative or toxic in character, would produce measurable variations in growth resulting from minimum seedings. Such mediums have been in use for some time.[5] The formula for the one chosen for our work is:

Composition of Medium by Weight (p_H 5)

Sucrose, C. P.	48.0	Gm.
Ammonium sulfate ($(NH_4)_2SO_4$)	3.0	Gm.
Potassium dihydrogen phosphate (KH_2PO_4)	4.0	Gm.
Calcium chloride ($CaCl_2.2H_2O$)	0.1	Gm.
Magnesum sulfate ($MgSO_4$)	0.25	Gm.
Casein extract	1.5	Gm.
Tryptophan	0.25	Gm.
Cystine	0.25	Gm.
Ferric chloride ($FeCl_3$)	0.10	mg.
Potassium iodide (KI)	0.10	mg.
Thorium chloride ($ThCl_4$)	0.10	mg.
Copper sulfate ($CuSO_4$)	0.10	mg.
Zinc sulfate ($ZnSO_4$)	1.0	mg.
Manganese chloride ($MnCl_2$)	1.0	mg.
Boric acid (H_3BO_3)	1.0	mg.
Distilled water	1000.0	ml.

5. Mosher, W. A., and others: Nutritional Requirements of the Pathogenic Mold Trichophyton Interdigitale, Plant Physiol. **11**:795-806, 1936. Williams, R. J., and Honn, J. M.: Role of "Neutrilites" in the Nutrition of Molds and Other Fungi, ibid. **4**:629-641, 1932.

For the preparation of the hair extract, preadolescent hair, referred to in the charts as 5 and 10 year hair, was obtained under supervision from an orphanage. The ages were approximate, within a year or so. The adult hair was obtained from an intelligently cooperative barber, and only hair from persons over 25 was used. Thus the extracts represent large groups of subjects and, in the case of the adult hair, a wide range of ages. After some consideration it was decided to limit the present study to water extract only. We came to this decision for several reasons. Primarily, many of the now known, so-called "growth substances" are definitely water soluble. In this group may be included such substances as vitamins B_1, B_2 and B_6, pantothenic acid and nicotinic acid. Even certain of the sterols, including some "sex hormones," while ordinarily not considered water soluble, are still sufficiently soluble to influence growth (if they are at all effective in that way) when their water extract is used. Finally, our choice of an aqueous "base" medium made the use of an aqueous extract more easily and accurately controlled.

PREPARATION OF HAIR EXTRACT

Preliminary work established the fact that it was preferable to work with fresh extract only. Apparently the extract is subject to deterioration. Similarly, hair which has been cut some time previously gives an extract of little use in the experiments. This may be due to the presence of toxic substances produced in the hair itself by autolysis. The experiments were carried on in each instance with small amounts of extracts, each freshly prepared from freshly cut hair. Our method was as follows:

Hair was collected in approximately 100 Gm. amounts and spread on a sheet of paper, and all possible foreign material was removed. After being thoroughly mixed, 10 Gm. was weighed and separated. This was chopped fine, thoroughly washed several times in cold water, drained and and placed in a liter beaker. To this 600 cc. of distilled water was added, and the mixture was vigorously boiled over a Bunsen burner, with mechanical stirring, for thirty minutes. The contents of the beaker were filtered, and the filtrate was evaporated on a steam table to approximately 10 cc. This concentrate was then transferred to a small previously weighed Erlenmeyer flask and evaporated to dryness. The flask was again weighed and the exact amount of concentrate determined. An amount of distilled water was then added to make a solution of 10 mg. of concentrate per cubic centimeter of solution. Any insoluble residue was removed by filtration, and the resultant solution was sterilized in the Arnold sterilizer on three successive days. It was then kept in the refrigerator until used.

THE SPORE SUSPENSION

Seeding has been done by means of water suspensions of spores. The preparation of these suspensions was unquestionably the most

difficult and at the same time the most important part of the experiment. One must recognize the possibility that, as with infection in man, a certain concentration (dosage) of the invading organism must be present for growth to result. At the same time, in studies of this type one can easily prove that if the so-called "dosage" is of considerable size, growths will result even in the presence of fairly large amounts of growth-deterrent substances. For the purpose of this study, therefore, it was highly important to use the minimum amount (dosage) of seedings. After considerable experimentation we attempted to accomplish this in the following way:

A portion of the mature growth on agar slants was removed by means of a platinum loop. This material was placed in a sterilized Erlenmeyer flask containing 10 cc. of distilled water. The flask was plugged with cotton, and a suspension was obtained by mechanical agitation for one hour. A uniform suspension, except with Microsporon fulvum, proved to be almost impossible to obtain. This fact, resulting in quantitative differences in seeding, apparently accounted for occasional inconsistent results. After agitation a sample was withdrawn and examined by means of a hemocytometer. By this means a final dilution was made approximating one spore to each 0.1 cu. mm. of suspension.

EXPERIMENTS

Erlenmeyer flasks of 50 cc. capacity, each containing 10 cc. of the base medium (previously described), were plugged with cotton and autoclaved for fifteen minutes at 15 pounds' (6.75 Kg.) pressure. After cooling, hair extract in portions of 2.5, 5, and in some cases 10 and 15 mg. was added. Into each flask 1 cc. of spore suspension was introduced. Controls not containing hair extract were made in each case. The flasks were kept at room temperature. Daily readings were made, and each experiment was terminated when the cultures in the medium containing extract of young hair had attained a well established growth. Organisms vary widely in their rate of growth under various conditions. Thus readings were made beginning in one or two days and continuing one or two weeks. In order to obtain the greatest possible accuracy, our first results were given in actual weights, which were obtained as follows: Gooch crucibles (fig. 1) of 1 cc. capacity were made. Into the stem was fused a finely perforated platinum plate, and on this was placed a fine asbestos filter pad. The apparatus was heated and weighed. Mass growths were removed from the flasks, thoroughly washed in distilled water to remove all traces of medium and transferred to the crucible, and the water was removed by suction. The growth was then thoroughly washed with ether and dried at 60 C. for thirty minutes. The crucibles and the residue were weighed and the weight of the growth residue computed.

In view of the time consumed and other conditions of the experiments not capable of being equally and accurately controlled, this method of estimating results was replaced by visual estimations in later tests. Each value represents a fair average of results obtained in series of from five to ten parallel experiments.

COMMENT

In table 1 are shown the average results obtained in a series of experiments with Microsporon audouini and M. fulvum. In the first column are shown the results of the controls. In the second column is noted the amount of hair extract added. In the remaining columns are the actual weights of the resultant growths after desiccation. When the growth was too small for weighing, it was recorded as a "trace." The growth of M. audouini was much greater in the presence of young hair extract than in the presence of mature hair extract or in the controls. M. fulvum showed definite growth in the controls, greater growth in

Fig. 1.—Gooch crucible used in obtaining actual weight of dried growths.

TABLE 1.—*Growth (Actual Weight) of Organisms in Medium Containing Extracts of Hair of Various Ages*

Control	Hair Extract Added	Weight, in Grams, of Growth of Microsporum Audouini After Desiccation		
		5 Year Hair	10 Year Hair	Mature Hair
Trace	2.5 mg.	0.0013	0.0008	Trace
Trace	5.0 mg.	0.0023	0.0020	Trace
Trace	10.0 mg.	0.0026	0.0024	Trace

Control	Hair Extract Added	Weight, in Grams, of Growth of Microsporum Fulvum After Desiccation		
		5 Year Hair	10 Year Hair	Mature Hair
0.005	5.0 mg.	0.0087	0.0085	Trace
0.005	15.0 mg.	0.0067	0.0064	Trace

the presence of young hair extract and a negligible amount in the presence of mature hair extract.

As noted previously, actual weighing of cultural growths necessitated a considerable amount of detailed work and prolonged the time of experimentation tremendously. After the foregoing experience it was believed that reasonably accurate, comparable values were obtainable by visual estimation. Thus, when there was a reasonably vigorous growth in one flask, that amount of growth was given a rating of 10 and the other growths were rated accordingly (fig. 2).

In table 2 are shown results obtained by this method. Included are the averages of a further series of experiments with M. fulvum and, in addition, experiments with M. lanosum. In the case of M. fulvum the results parallel fairly closely those obtained by actual weighing. Moderate growth occurred in the controls, greater growth in the presence of the extract of young hair and no growth in the presence of the extract of mature hair. In the case of M. lanosum no growth

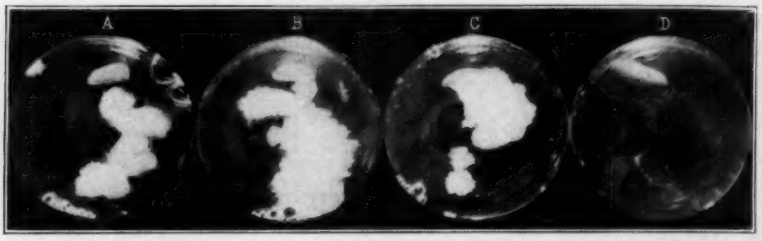

Fig. 2.—Growths from which visual estimations were made, *A*, control, *B*, prepuberty hair (5 years), *C*, prepuberty hair (10 years) and *D*, mature hair. Photograph obtained by placing flasks containing growths on plate glass and pointing the camera upward from below. Thus the photographs actually show only the base area of the growths.

occurred in the controls or in the mediums containing the extract of mature hair. Definite growth occurred in the presence of extract of young hair.

TABLE 2.—*Visual Estimation of Growth of Organisms in Medium Containing Extracts of Hair of Various Ages*

		Rating of Average Visual Growth of Microsporum Fulvum		
Control	Hair Extract Added	5 Year Hair	10 Year Hair	Mature Hair
5	2.5 mg.	10	8	0
5	5.0 mg.	10	8	0
		Rating of Average Visual Growth of Microsporum Lanosum		
Control	Hair Extract Added	5 Year Hair	10 Year Hair	Mature Hair
0	5.0 mg.	10	5	0

It is interesting to note that M. audouini grew poorly in the synthetic medium used, that M. fulvum grew only fairly well and that M. lanosum showed no growth at all. In part this is explained by the differences in nutritional requirements of the various pathogenic fungi. A further explanation is presumably furnished by the minute seedings employed. For the purposes of this investigation, however, it seemed that only a medium sensitive to extremely small amounts of nutritional or toxic substances should be considered. The amount of growth in the presence of extract of young hair was not in proportion to the amount of extract added. For this we have no explanation. The use of smaller or larger amounts might furnish the answer. The results with the mature hair extract are striking. From time to time during the study occasional inconsistencies occurred. For the most part they seemed explained by inaccuracies in seeding or the use of extract that had stood too long. In the charts the perfect results are not shown, nor are the glaring inconsistencies. The charts represent the average results obtained.

SUMMARY

In the foregoing are detailed the results of experiments undertaken in an effort to throw some light on a common, unexplained clinical observation, i. e., the common occurrence of ringworm of the scalp in children and its infrequency later in life. The results noted, together with those of an extended period of preliminary experimentation, are only partially conclusive. To those acquainted with this type of investigation the impossibility of accurate control of certain procedures is familiar. Thus occasional inconsistent results are neither surprising nor disappointing. Unquestionably the work should be extended to include ether, alcohol and perhaps other hair extracts. The possible influence of "hormones" should be investigated. Furthermore, a larger group of organisms should be included in this and other methods of approach. With full appreciation of the information still lacking, it would seem that in the foregoing text has been demonstrated a difference in hair of different ages which may be a definite part of the final explanation of the question under discussion.

Under the conditions of these experiments, seedings of Microsporon audouini, M. fulvum and M. lanosum grow more abundantly in a medium containing extract of young hair than in a medium containing extract of old hair or no extract at all. This might be explained by the presence in young hair of some substance having nutritional properties or in old hair of substances with inhibitive or toxic properties.

ABSTRACT OF DISCUSSION

DR. DONALD M. PILLSBURY, Philadelphia: I have been interested in the subject of tinea capitis somewhat by compulsion, because of the large number of cases seen at the clinic of the Children's Hospital. Some years ago Dr. James Andrews,

of the department of physiologic chemistry of the University of Pennsylvania, prepared a complete hydrolysate of adult hair for me, and I treated several patients with tinea capitis by the application of the hydrolysate in liquid and in ointment form, in a concentration considerably in excess of that used by Dr. Kingery and his co-workers in their experiments. I did not note any significant influence of this treatment on the disease, but my clinical experiment was admittedly crude and obviously of no value in comparison to the precise laboratory studies which Dr. Kingery and his co-workers have presented.

As far as I know, this is the first attempt to explain the interesting phenomenon of tinea capitis ordinarily disappearing at puberty. It is difficult to analyze charts shown for a moment or two, but Dr. Kingery and his collaborators seem definitely to have demonstrated a variation in the growth of fungi when they are exposed to some substance extractable from adult hair as compared to that extractable from preadolescent hair. In this connection, I should like to inquire whether the method of collecting the hair reasonably excluded the possibility of some extraneous substance, such as hair tonics, soap, etc., which might possibly be present in greater concentration in adult hair than in preadolescent hair, though this seems unlikely.

Dr. George M. Lewis, New York: Methods of therapy for tinea capitis may improve when the reason for the vulnerability of the child's scalp and the high degree of resistance of the adult's scalp is known. Dr. Kingery and his associates have investigated a phase of this problem, with rather striking results. There is evidence that spontaneous cure of tinea capitis at puberty may be due to: (1) an immunologic or sensitization phenomenon or (2) alteration in the character of the hair follicle due to an increased titer of estrogenic hormones in the body fluids. The concept of an immunologic or sensitization basis for cure is strengthened when one notes that a follicular inflammatory process resulting in epilation is the apparent reason for the occasional spontaneous cure at an earlier age and for cures which result from the application of medicaments. In such cases a positive reaction to the trichophytin test is an associated finding. I recently observed a Negro girl of 14 with tinea capitis in whom the menses had been established one year previously. The infection had been present for six or more years. It was masked by a vigorous growth of normal hair. Cultures showed Microsporum audouini. The trichophytin test gave a negative reaction. At present a biologic assay of estrogenic hormone is being carried out. I hope to be able to follow this case, which is rather unique in my experience and I think fairly uncommon in the general run of experience.

Dr. Lyle Boyle Kingery, Portland, Ore.: Dr. Pillsbury has brought up a question to which we gave a great deal of thought. We finally decided, however, to carry on as described. We took the hair samples as they came, subjected all to the same thorough washing and made sure that each sample of hair extract used included hair from a large number of persons.

NECROBIOSIS WITHOUT DIABETES

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Since the original description of necrobiosis lipoidica diabeticorum, many cases of the disease have been presented. While this entity still needs some elucidation as regards genesis, practically every one professing a special knowledge of dermatology recognizes it clinically.

During the past few years there have been mentioned in the literature several cases in which the clinical observations suggested the aforementioned entity but the patients did not have diabetes mellitus. The first of these was recorded by Goldsmith,[1] and while his clinical diagnosis favored a morphea-like tuberculosis, microscopic studies pointed toward necrobiosis lipoidica. Gross [2] presented a case as one of "morphea with xanthomatous infiltration," but more recently he has referred to it as one of necrobiosis without diabetes.[3] This case was originally presented before that of Goldsmith but was not recognized as such. In both the foregoing instances diabetes mellitus was ruled out by dextrose tolerance tests and determinations of blood cholesterol, which showed normal levels. More recently cases have been recorded by Greenwood and Rockwood [4] and by Bruce-Jones,[5] and we wish to add

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Hospitalization and study of this patient were made possible by a grant from the Horace H. Rackham School of Graduate Studies.

Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 2, 1939.

Studies and contributions from the Department of Dermatology and Syphilology of the University of Michigan Medical School; service of Dr. Udo J. Wile.

1. Goldsmith, W. N.: Necrobiosis Lipoidica, Proc. Roy. Soc. Med. **28**:363 (Feb.) 1935.

2. Gross, P.: Morphea with Xanthomatous Infiltration, Arch. Dermat. & Syph. **30**:598 (Oct.) 1934.

3. Gross, P., in discussion on Vero, F.: Necrobiosis Lipoidica Diabeticorum, Arch. Dermat. & Syph. **39**:733 (April) 1939.

4. Greenwood, A. M., and Rockwood, E. M.: Necrobiosis Lipoidica Diabeticorum [case 7], Arch. Dermat. & Syph. **35**:727 (April) 1937.

5. Bruce-Jones, D. B. S.: A Case Clinically Resembling Morphea with a Tuberculous Background and Indeterminate Histology Suggestive of Necrobiosis Lipoidica, Brit. J. Dermat. **49**:238 (May) 1937.

a fifth,[6] clinically indistinguishable, in which diabetes mellitus has been excluded.

REPORT OF A CASE

History.—A. A., a white housewife aged 28, first appeared for examination on Jan. 25, 1937, with the complaint of an eruption confined to the legs. The original lesion had appeared seven or eight years previously and was described as a "small red spot" below the left knee, overlying the shin. She was unable to recall any preceding or subsequent trauma to this area. This lesion remained asymptomatic but increased in size slowly. In 1936 she first noticed scaling of the surface and a purple "halo" around the lesion. A month or two before coming

Fig. 1.—Distribution of the cutaneous lesions.

to the hospital a definite yellow color in the central portion was noted. During the previous two years two new lesions, similar in appearance, had appeared: the first on the right shin and the other on the left leg just mesial to the original lesion. These followed the same progressive course. Treatment with various salves, including a coal tar preparation, and ultraviolet radiation had been entirely ineffective.

The history of the patient and of the family revealed no traces of diabetes, tuberculosis or syphilis and were otherwise not significant.

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6. This case has previously been presented at meetings of the Chicago Dermatological Society, Arch. Dermat. & Syph. **36**:912 (Oct.) 1937, and the American Academy of Dermatology and Syphilology, Arch. Dermat. & Syph. **38**:311 (Aug.) 1938.

Physical Examination.—On admission the patient was well developed and well nourished and had three lesions of the skin which were confined to the anterior aspect and upper third of the legs. The largest lesion, just below the left knee, measured 3.8 cm. in diameter and appeared as a round, slightly irregular plaque, the central portion of which presented a definite yellow color, more evident on diascopic examination, with superimposed telangiectasia, while the peripheral portion appeared dully erythematous with a violaceous halo. The surface was covered

TABLE 1.—*Blood Sugar Levels* *

Date	Fasting Level, Mg./100 Cc.	First Hour, Mg./100 Cc.	Second Hour, Mg./100 Cc.	Third Hour, Mg./100 Cc.	Fourth Hour, Mg./100 Cc.
1/27/37	83	84	65	59	..
12/ 3/3	64	68	65	51	..
2/ 8/38	78	..	..	..	..
2/ 9/38†	89	..	..	..	..
2/10/38‡	75	81	91	74	57
2/11/38‡	72	..	..	..	..
2/12/38	58	..	..	..	..

* Hourly urine specimens during each dextrose tolerance test were all negative for sugar.
† The patient was on a high carbohydrate (weighed) diet on Feb. 7, 8 and 9, 1938 (in hospital).
‡ The patient was on a fasting diet (tea, lemon, coffee with cream, and water).

TABLE 2.—*Blood Lipoids*

	Total Lipoid, Mg. per 100 Cc.	Cholesterol, Mg. per 100 Cc.			Phospholipid, Mg. per 100 Cc.
		Total	Esters	Free	
Method	Eckstein gravimetric (serum)	Sackett's modified Bloor (whole blood)	Schoenheimer and Sperry		Fisk and Subarrow (serum)
Our case:					
Normal range or aver...	400 to 700	150 to 200	160	120	40	150 to 200
Feb. 10, 1937	660	190	...	...	..	...
March 12, 1937	...	178*	...	...	..	...
March 3, 1939†	493	...	174.5	134.5	40	189
Case of Greenwood and Rockwood (stated normal)	700	250	160	70	Phospholipid-phosphorus 9 to 10
Case of patient (C. M.)...	725	269	195	74	9.8
Case of Goldsmith	...	150			
Case of Gross	...	173			

* This value was obtained four hours after a cholesterol ingestion test (Burger and Habs).
† Dr. H. C. Eckstein, of the department of biological chemistry, made these determinations as well as the tissue analysis.

by a thin whitish superficial scale. The other two lesions, smaller in size, showed the same general characteristics. There was no tenderness on pressure, and none of the lesions showed ulceration. Examination in the ophthalmology department showed normal fundi and normal visual fields. The remainder of the physical examination gave essentially negative results except that the liver was just palpable and the spleen questionably enlarged.

Clinically the cutaneous lesions presented all the characteristics of necrobiosis lipoidica diabeticorum, and consequently a thorough investigation by pertinent laboratory methods was carried out in an attempt to find or to rule out diabetes of either type and any disturbance of the blood lipoids.

Laboratory Data.—The blood on admission showed hypochromic anemia, which responded rapidly to reduced iron administered by mouth, to a point of temporary polycythemia; the red cell count soon became normal and has remained so without any additional iron therapy. At one time there was also a temporary increase in the percentage of monocytes (10 per cent).

Complete examination of the urine gave negative results, and many repeated tests have never revealed a trace of albumin or sugar. The basal metabolic rate

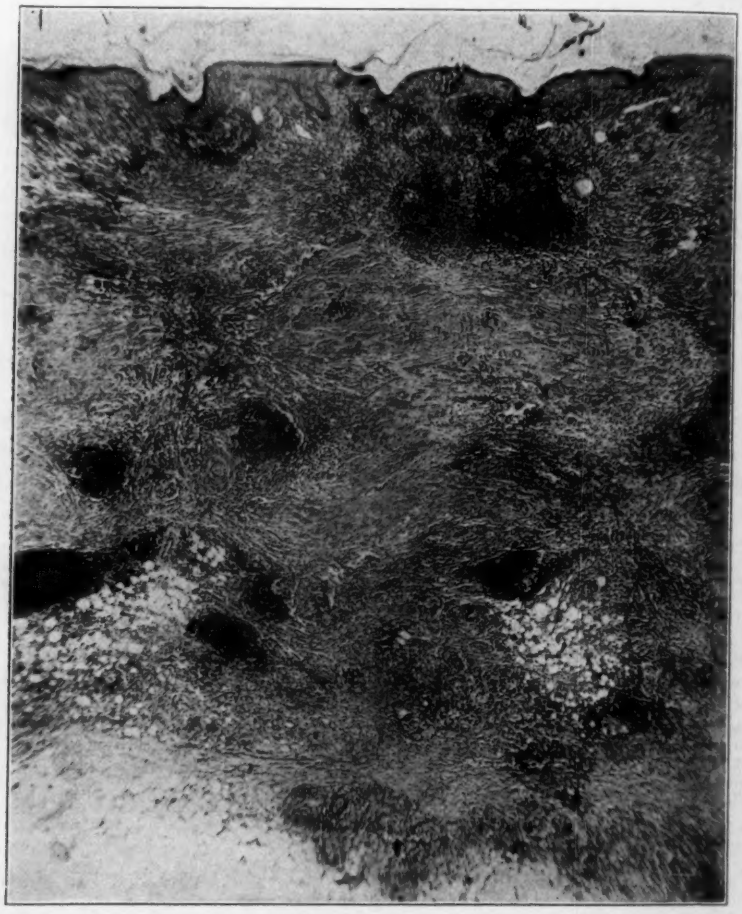

Fig. 2.—Atrophy of the epidermis, the focal character of the infiltrate and decided changes in the collagen (low power).

was —5 per cent on Feb. 8, 1938, and —3 per cent on February 9. The Kahn test of the blood has been repeatedly negative.

Roentgenograms of the skull showed no evidence of intrasellar erosion or of any intracranial or cranial disease. On roentgenologic examination, the chest, including the heart, aorta, diaphragm and thoracic cage, appeared normal. The lung parenchyma was clear, with no evidence of intrathoracic disease.

Dextrose tolerance tests were carried out on three separate occasions, the results of which, along with the blood sugar levels during fasting as determined on four other occasions, appear in table 1.

The results of blood lipoid determinations appear in table 2, along with those reported by Greenwood and Rockwood [4] and Goldsmith.[1] Blood carotinoids were in the upper normal range (White and Gordon method).

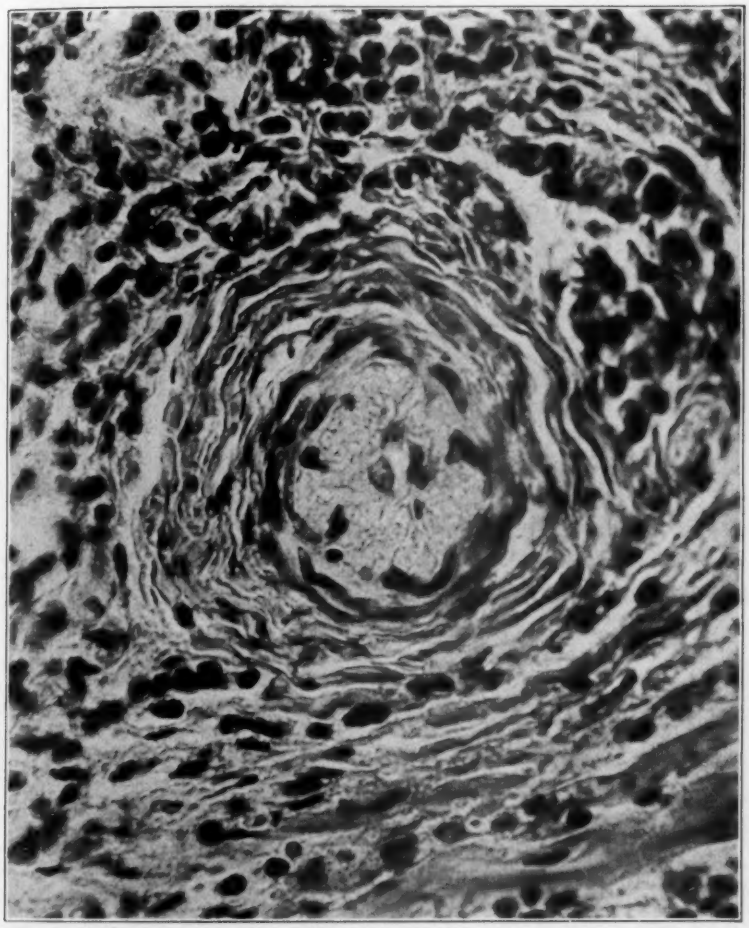

Fig. 3.—Marked vascular change, consisting of proliferation of the intima, and almost total occlusion of the lumen (high power).

Microscopic Study.—Biopsy specimens were taken at intervals over a period of two years, and, except in degree, the changes were the same. In sections stained with hematoxylin and eosin, the epidermis showed mild hyperkeratosis and parakeratosis and, in addition, a decided flattening of the rete pegs, which probably represented atrophy. In the upper layers of the corium there was some dilatation of the vascular spaces and, in addition, a diffuse mononuclear infiltrate. Deeper in the corium there was patchy necrosis of the collagen and elastic fibers,

which appeared particularly as a homogenization with a distinctly bluish tinctorial change. Associated with these areas were focal infiltrates of lymphoid and plasma cells and occasional multinucleated giant cells. Close to the focal infiltrates the small vessels showed distinct damage, evidenced by thickening and swelling of the intima and in some cases practical occlusion of the lumen.

Scarlet red in frozen sections showed some clumps of stainable fat scattered extracellularly through the tissue, although they were not present in all areas.

Fig. 4.—Distribution of lipoid.

In addition there was some fat contained within the phagocytic cells. The giant cells were distinctly not the Touton type, and no typical foam cells could be found.

Course.—During the twenty-eight months this patient has been under periodic observation, the original lesions have slowly but steadily increased in size, and two new lesions have appeared, one of these only one month ago. No ulceration has occurred in any of the lesions, and they have all remained asymptomatic. After the last biopsy there appeared to be a temporary decrease in the intensity of the yellow color, but it returned to its former state soon after the wound for removal of the biopsy specimen healed. A course of superficial roentgen treatments started recently has had absolutely no effect so far on the nature or progress of any of the lesions.

COMMENT

When we reviewed the clinical findings, we were faced with surprisingly little evidence of disease: only the palpable liver and the obvious cutaneous lesions. The laboratory studies added little of importance. The blood lipoids were well within the normal range, and this was approximately true also for the other cases of necrobiosis without diabetes for which figures have been reported (table 2). While the case of Greenwood and Rockwood [4] showed some slight increase, we doubt that it was sufficient to be significant.

A comparison with blood lipoid values in cases of true necrobiosis lipoidica diabeticorum was difficult because complete figures were not often reported, and the methods used in determining normal values were seldom stated. A survey of about 25 reported cases showed a definite elevation of total lipoids in 2 out of 4 and an increase of the total cholesterol in 9 out of 17.

Tissue excised from the largest lesion showed, after the subcutaneous fat had been teased away, a total lipoid content of 3.4 per cent and,

TABLE 3.—*Tissue Analysis*

Total lipoids, percentage of total material (gravimetrically)	3.4
Total cholesterol*	
Percentage of total material	0.34
Percentage of total lipoids	10.0

* This was determined by the Bloor colorimetric method.

of this, a total cholesterol content of 10 per cent (table 3). Because of the small amount of tissue allowed us, we were not able to break this down into free cholesterol and cholesterol esters. Cases of true necrobiosis lipoidica diabeticorum have a tendency to show increased total lipoid values in tissue excised from the lesions, as exemplified by the case of Usher and Rabinowitch.[7]

One other lipoid study in our case may be of some importance. Table 2 shows that there was no elevation in blood cholesterol after a cholesterol meal. In some quarters this is regarded as evidence of liver damage, and, when we add to this the fact that our patient had a palpable liver, it may be significant.

The other laboratory procedure requiring comment is the dextrose tolerance test. When we found repeatedly a low, flat type of curve and a considerably lower level of blood sugar after a fasting diet, we felt that we had evidence pointing toward pituitary disease. However, it now appears that this type of response is normal for young persons,

7. Usher, B., and Rabinowitch, I. M.: Necrobiosis Lipoidica Diabeticorum, Arch. Dermat. & Syph. **35**:180 (Jan.) 1937.

and in the near future Conn[8] will publish evidence that this is true. When we considered with this the normal basal metabolic rate, the normal ocular fundi and visual fields and the normal roentgenogram of the skull, we were forced to abandon the diagnosis of pituitary disease.

While many conditions might be considered in a differential diagnosis, it appeared to us that tuberculosis, xanthoma, extracellular cholesterosis and necrobiosis lipoidica diabeticorum possessed features most similar to those of our case.

It is interesting to point out that the cases of Goldsmith[1] and Bruce-Jones[5] suggested an unusual morphea-like indurative tuberculosis, and indeed the first case was presented with that clinical diagnosis. Only microscopically was the condition diagnosed necrobiosis lipoidica. Gross's case,[2] presented originally as one of morphea with xanthomatous infiltrate, must have been similar from the clinical standpoint. In our case the original diagnosis was necrobiosis lipoidica, and in the early biopsy the evidence was all in favor of this diagnosis. Subsequent biopsies have shown, in addition to the necrobiotic foci, a distinct tendency toward a more granulomatous change, which has led Prof. Carl V. Weller, of our pathology department, to raise the question of an unusual form of tuberculosis. In view of this highly regarded opinion we hesitated to close the door to this as a possibility. However, the localization, the distinct yellow color, the type of lesion and the failure to demonstrate evidence of the usual or sarcoid type of tuberculosis elsewhere made this diagnosis unlikely.

Largely because of the distinct yellow color of the lesions, xanthoma had to be considered. This, however, was about as far as the likeness went. The distribution was unusual; no typical foam cells could be found, and the giant cells were not of the Touton type. In addition, the total lipoid content of the tissue in our case was close to the upper limit of normal (3.4 per cent), whereas the total lipoid in xanthomatous tumor tissue is commonly three or four times that amount.[9] One might possibly raise the question of xanthoma areolare multiplex of Riehl and Arzt.[10] However, this condition is characterized by widely disseminated, sometimes verrucous tumors, which have a tendency to ulcerate, a deep brown areola being seen around each lesion, and by a decided regional adenitis. In addition, foam cells are described.

Considered clinically, the condition under discussion has absolutely nothing in common with extracellular cholesterosis. As described by

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8. Conn, J. W.: Personal communication to the authors.

9. Wile, U. J.; Eckstein, H. C., and Curtis, A. C.: Lipid Studies in Xanthoma, Arch. Dermat. & Syph. **19**:35 (Jan.) 1929. Eckstein, H. C., and Wile, U. J.: Lipid Studies in Xanthoma, J. Biol. Chem. **87**:311 (June) 1930.

10. Arzt, L.: Beitrag zur Xanthom-Frage, Arch. f. Dermat. u. Syph. **126**:809 (April) 1919. Urbach.[11]

both Urbach [11] and Laymon,[12] this condition consists of widespread polymorphous lesions, and outstanding among them are distinct tumors of reddish brown rather than yellow color. In Urbach's case the tumors were verrucous, while in Laymon's case the tops of the lesions were described as smooth. The outstanding likenesses were the evidence of hepatic damage, the evidence of vascular damage and, for the most part, extracellular deposition of lipoid. While the lack of giant cells has been stressed in extracellular cholesterosis and many were found in our case, we do not like to place too much importance on this as a differential point, because we think it an unsafe one.

Both clinically and microscopically the condition we have described approached necrobiosis lipoidica diabeticorum more closely than any other condition with which we are acquainted. But there were some differences. Neither diabetes mellitus nor diabetes insipidus was present. The early reports stressed the absence of giant cells. This, however, appears without significance since Michelson [13] and Hitch,[14] among others, have since reported apparently typical cases in which they appeared. In the true cases apparently the lipoid is abundant and easily found throughout the sections, whereas we had considerable difficulty in demonstrating unusual stainable fat. Finally we found the lipoid content of both tissue and blood within the normal range, while cases in which there was diabetes have for the most part shown some significant elevation.

SUMMARY

We present here a condition in which there is evidence of hepatic damage together with cutaneous lesions of a xanthocromic type which microscopically show vascular damage, focal necrobiosis and deposition of both extracellular and intracellular lipoid. Because it approaches necrobiosis lipoidica diabeticorum more closely than any other condition with which we are acquainted, we call it necrobiosis without diabetes. We believe it is another of an accumulating group of lipoid disturbances which do not quite fit into the present classification. If we are justified in this belief we are forced to conclude that, while necrobiosis lipoidica probably affects persons with diabetes more commonly than others, it is not necessarily a part of, or dependent on, diabetes mellitus.

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11. Urbach, E.: Lipoidstoffwechselerkrankungen der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1932, vol. 12, pt. 2; Geschwülste der Haut, ibid., pt. 1, p. 238.

12. Laymon, C. W.: Extracellular Cholesterosis, Arch. Dermat. & Syph. **35**:769 (Feb.) 1937.

13. Michelson, H. E., and Laymon, C. W.: Necrobiosis Lipoidica Diabeticorum, Arch. Dermat. & Syph. **35**:1130 (June) 1937.

14. Hitch, J. M.: Necrobiosis Lipoidica Diabeticorum (Urbach and Oppenheim), Arch. Dermat. & Syph. **36**:536 (Sept.) 1937.

ABSTRACT OF DISCUSSION

DR. EUGENE F. TRAUB, New York: Dr. Belote and Dr. Welton's paper interested me because of 3 cases which I presented at various meetings during the past five years. The first case was presented at the April 1934 meeting of the New York Dermatological Society (ARCH. DERMAT. & SYPH. **30**:902 [Dec.] 1934) and the May 1934 meeting of the New York Academy of Medicine and subsequently was shown before the clinical meeting of the American Dermatological Association in June 1934. The case was presented as one for diagnosis (morphea-like tuberculosis cutis?). Necrobiosis lipoidica diabeticorum was considered, but because of the absence of diabetes and certain other laboratory findings and because of the mode of onset I thought this diagnosis could be excluded. Drs. Wile, Sweitzer and Montgomery, who discussed the case, thought that the condition fitted in with the clinical picture of necrobiosis lipoidica diabeticorum. After studying the case further and seeing 2 similar cases, I decided that this was the correct diagnosis despite the definite absence of diabetes. Extensive cholesterol studies as well as sugar tolerance curves and blood sugar tests were reported. Three or four sections of tissue had also been removed for microscopic study, and peculiarly enough the area where the tissue had been removed healed exceptionally well, leaving a thin white flexible scar quite different from the original lesion and almost approaching the normal skin in texture.

The second case to which I refer was that of a Norwegian woman, S. G., 34 years of age, presented at the New York Dermatological Society meeting in October 1936 (ARCH. DERMAT. & SYPH. **35**:1194 [June] 1937). She was presented for diagnosis (morphea-like lesions with a tuberculoid structure?). Again a diagnosis of necrobiosis lipoidica diabeticorum was considered, but Dr. Frank Fraser thought that the characteristic necrotic masses were absent when he examined the microscopic slides; therefore, since the patient did not have diabetes and since Dr. Fraser thought the section showed the structure of a xanthoma, the case was not recorded as one of necrobiosis lipoidica diabeticorum.

The third case was identical with the first two mentioned, and it was not until I had observed this case that I realized that the 3 cases belonged in a separate category, or under a classification of necrobiosis lipoidica diabeticorum without diabetes. The case was presented at a hospital meeting and is not recorded in the literature.

All my patients were followed for a period of years, especially the first one, who was studied for three or four years until she left the city. Of course, Dr. Belote and Dr. Welton could not include these cases in their series because of the way 2 of them were recorded in the literature and because 1 was not recorded, but that they belong in this group there is no doubt.

A few days ago I saw a patient who had been operated on recently for extensive lupus vulgaris of the scalp and body. In view of the fact that the tissue examination in my cases suggested to many of the dermatologists a morphea-like lesion with a tuberculous or tuberculoid structure, the case I am about to describe may be significant from two angles. The patient, who was in her late forties, had a tremendously large Thiersch graft placed over the greater part of her scalp after the lupus tissue was excised. The entire graft took well, and the new area of skin appeared to be normal for about eight or nine months, at which time a change occurred, which if seen on the leg could easily have been confused with a classic picture of necrobiosis lipoidica diabeticorum. This patient did not have diabetes, but it is possible that the graft placed on the skull may have been subjected to certain nutritional changes due to the relatively poor circulation in

that area and hence possibly poor nourishment—that is one possibility. The other possibility is that the tuberculosis might have been a factor in bringing about this peculiar waxy, yellowish change in the Thiersch graft, together with the increase of the superficial vessels. This case may be of real value in tracing the causation of necrobiosis lipoidica diabeticorum without diabetes.

Dr. Erwin P. Zeisler, Chicago: Dr. Belote and Dr. Welton have presented a characteristic case of necrobiosis both clinically and histologically. It conforms to the cases Dr. Caro and I reported several years ago. They have also proved that the patient did not have diabetes at the time these tests were made. He may have had diabetes prior to that time. There may also be cases of necrobiosis in which clinical diabetes develops later. Even these occasional cases reported without evidence of diabetes will not militate against the fact that the clinical picture in the majority of cases shows diabetes and in most cases severe diabetes. I should like to mention one or two details that differ from some of the reported cases. In the histologic picture I did not see the grapelike arrangement of extracellular lipoids in the tissue reported in some cases. The concentric involvement of the blood vessels, which indicates severe vascular damage, is reminiscent of the findings reported by Balbi in the Italian literature. Dr. Belote and Dr. Welton said nothing about therapy, so that I do not know whether the patient was treated. Dr. Caro and I have had our original patient under observation for many years. He still has severe diabetes, and in spite of continuous insulin therapy the cutaneous changes are still present. There is slight atrophy, but the lesions still have a yellow color and some are slightly indurated. I believe, therefore, for all practical purposes that the disease is chronic and incurable.

Dr. Donald M. Pillsbury, Philadelphia: I should like to record a case which I recently observed and in which the diagnosis of necrobiosis lipoidica diabeticorum was confirmed by Dr. Eric Urbach. This patient, a physician, had no evidence of diabetes; in fact, his dextrose tolerance curve was of a type almost suggestive of hyperinsulinism. The blood cholesterol was normal, but determination of various lipoid fractions was not done. It would seem that the evidence is increasing that this syndrome is not necessarily associated with diabetes. It seems also possible that cases exist in which there is no general disturbance of the lipoid metabolism, at least not by the methods available for determination at the present time. The lipoid infiltrates observed in the lesions might possibly be only a local change and not certain proof of a general disturbance in this regard; at least this interpretation seems worthy of consideration.

Dr. Arthur M. Greenwood, Boston: It has seemed to me that the type of case which Dr. Belote and Dr. Welton have reported may possibly offer a solution to one of the great problems in the treatment of diabetes. Persons with diabetes do not die from diabetes any more but from complications, such as injury to arteries particularly. It is agreed that injury to the blood vessels is found alike in cases of necrobiosis with diabetes and in cases of necrobiosis without diabetes. It is agreed also that disturbance of the lipoid metabolism is found in both types of cases. There are then these two different types of necrobiosis, one associated with diabetes and one without diabetes. The patients with diabetes show disturbance of both the lipoid and the carbohydrate metabolism and injury to the blood vessels. Those without diabetes show injury to the blood vessels and altered lipoid metabolism. Can one then eliminate the diabetogenic factor of the anterior pituitary lobe in considering the problem of injury to the blood vessels? The problem is a complicated one, and if one can by these cases eliminate the possibility of the influence of the anterior pituitary lobe one has eliminated one complicating factor.

I have seen 12 cases of necrobiosis with diabetes and but 1 case without it. In no case of fully developed lesions was there any question of the diagnosis. It is in cases in the early stage that difficulty arises so far as diagnosis is concerned. Early lesions show practically no resemblance to those that later develop. In some of my cases, 1 in particular, there was no development of characteristic lesions for eight or nine years. That diagnosis was proved microscopically.

Dr. Marion B. Sulzberger, New York: I believe that one may today perhaps consider certain disturbances of lipoid metabolism as partial diabetes. In other words, diabetes consists not only of a disturbance of the carbohydrate metabolism but of a disturbance of the lipoid metabolism. Perhaps the part of the diabetes which usually causes the lesion of necrobiosis diabeticorum is not the disturbance of the carbohydrate metabolism itself but the accompanying disturbance of the lipoid metabolism. That brings up the point stressed by Schaaf in his recent experimental work on the metabolism of lipoids (*J. Invest. Dermat.* **1**:11, 1938). Schaaf showed fairly conclusively that one of the important functions of the liver is keeping the lipoids in emulsion. According to Schaaf's results, it seems that the liver regulates the ratio between cholesterol, cholesterol esters, free fatty acids and other lipoids in the blood, so that stable emulsions are formed. Therefore, when this function of the liver is impaired, as in certain forms of hepatic disease, one is likely to find deposits of lipoids in any area of damaged tissue. In Dr. Belote and Dr. Welton's case there was evidence of hepatic damage. It is therefore possible that in this case there was present just that part of the symptom complex of diabetes which leads to faulty fat metabolism. The basis of necrobiosis lipoidica diabeticorum may be a form of hepatic damage; and this damage and faulty emulsification of fats may occur with or without other manifestations of diabetes.

Dr. Henry D. Niles, New York: May I be permitted to add 1 more case? The patient was a middle-aged woman, a private patient. I did not report this case because I was unable to study it completely. From the endocrine point of view, the interesting feature was that the lesions appeared after hysterectomy and removal of one ovary. Clinically, there were typical patches varying from the size of a lentil to that of a bean; they had scleroderma-like, yellow, waxy, atrophic centers, covered with telangiectatic vessels, and violaceous borders. Histologic examination showed areas of necrosis and diffuse collections of giant, round and epithelioid cells. Unfortunately it was impossible to make special fat stains. There was no evidence of diabetes. The blood sugar content was 88.9 mg. per hundred cubic centimeters, the cholesterol content 111.1 mg. This patient was put on a low fat diet and given ¾ grain (0.0486 Gm.) of thyroid daily. She has been followed for some time, but there has been no change in her eruption.

Dr. Louis A. Brunsting, Rochester, Minn.: It has been my experience in about a half dozen of these cases that all were associated with diabetes. The question of diabetes cannot be settled with certainty by a limited period of observation, especially in young persons. Other factors may be necessary for the full development of diabetes, particularly the approach of middle age and a gain in weight. The general discussion of cases of necrobiosis lipoidica diabeticorum by dermatologists has had a salutary effect on the broadening of the specialty in relation to internal medicine. On a number of occasions recently it has been my experience to have internists ask for consultation about pigmentary disturbances of the ankles and other lesions of the lower portion of the legs in cases of disturbed carbohydrate metabolism to decide whether such changes were evidence of necrobiosis.

Dr. M. H. Ebert, Chicago: I wish to put on record a case through the courtesy of Dr. Oliver S. Ormsby. The patient was a woman of 42 who had

typical lesions of necrobiosis lipoidica diabeticorum. They had been present for several years without any apparent change. We had but a brief opportunity to study her but did have some opportunity to study the dermatitis. The interesting feature was that there was no evidence of diabetes. The blood sugar on two occasions was normal; the sugar tolerance test was normal, but the total blood lipoids were double the normal amount. In this case I should be inclined to think that there was some definite disturbance of the lipoid metabolism, as in some of the cases of xanthoma and xanthelasma reported by Dr. Montgomery. I was inclined to believe that there was involvement of the cardiovascular system, but none was discovered clinically. I think that every one realizes that in diabetes there is a disturbance not only of the carbohydrate metabolism but of the lipoid metabolism. Some of these cases of necrobiosis belong to the latter class.

Dr. Fred D. Weidman, Philadelphia: There seems to be a consensus that the condition in this case conforms at least clinically to necrobiosis lipoidica diabeticorum; therefore, all I want to speak about is the tissue process—the pathologic change. The necrobiosis is of the order which is seen in a wide range of arteriosclerotic conditions. In infarcts, for example of the heart, necrobiotic foci occur; and in infarction of the spleen, kidney and lungs there are actual necroses with a cone-shaped contour. The latter form is omitted in the skin in this case because of the differing distribution of the blood vessels; where arteries are terminal, as in the liver, spleen and lungs, the cone-shaped lesion is easily understood. It is well established that in the necroses which accompany infarction there is also fatty degeneration of tissue, and this could account in part for the fatty features of necrobiosis lipoidica diabeticorum. Incidentally, in infarction there may also be disturbances in the lipoid content of the blood, because the arteriosclerosis which can cause infarction is frequently accompanied by hypercholesteremia. As a matter of fact, in some cases of xanthoid "tumors" of the shins there have been yellowing changes produced independently of any systemic disturbance of the lipoid metabolism in which the yellow color appeared to be due to necrosis as well as to blood pigmentation. Broders, for example, has reported benign extraperiosteal "tumors" of the leg which were probably only traumatic in origin. Of course, in diabetes mellitus there is notoriously arteriosclerosis, which is ample explanation why necrobiosis may occur in the legs.

In this case, however, in which there has been failure to demonstrate carbohydrate abnormalities, one must turn somewhere else. I would suggest that perhaps this is the end stage of some preexisting acute vascular condition. It might be well for one to keep this case in mind when Dr. Ketron and Dr. Bechet read their papers on periphlebitis nodularis necroticans and on periarteritis nodosa. One should try to think of some essential disease of the arteries such as these conditions in order to explain all cases of necrobiosis, whether associated or not with diabetes mellitus.

Dr. George H. Belote, Ann Arbor, Mich.: I have one question that I wish to try to answer, that of Dr. Zeisler. The question of preexisting diabetes has been brought up on several occasions. I cannot say that this patient did not have diabetes ten years ago when the condition started; no one could make that assertion at the present time. Dr. Welton and I have had her under observation for two and a half years, and we have seen new lesions develop clinically, but she does not have diabetes in the ordinary sense of the word.

PERMEABILITY AND ABSORPTIVITY OF THE SKIN

JOSEPH J. ELLER, M.D.
AND
SHIRLEY WOLFF
NEW YORK

Because of the systemic and local effects of the cutaneous application of various medicaments, the permeability and absorptivity of the skin are of particular importance to the dermatologist.

As far back as 1809 Mussey [1] proved that after topical application of certain drugs to the unbroken skin these drugs were found in the urine, and in 1901 Reilly [2] reported therapeutic effects after cutaneous application of such substances as belladonna, mercury, pilocarpine and cod liver oil and also the presence in the urine of salicylic acid, turpentine, guaiacol, creosote and phenol.

It has been demonstrated [3] that substances soluble in oil are more readily absorbed through the unbroken skin than those soluble in water. Zondek [4] reported that drugs in solvents such as alcohol, ether, benzine and chloroform are more rapidly absorbed and in greater amounts than those incorporated in an oily base. He also reported that estrogenic substances in alcoholic solution were percutaneously absorbed more rapidly than those in an oily vehicle. How the vehicle influences the rate of absorption or penetration is still to be determined. This is an extremely difficult problem, since many physicochemical factors are involved.

Macht [5] found that most essential or volatile oils, such as cinnamon, wintergreen, cloves, peppermint, lemon and orange, are readily absorbed through the intact skin. He reported the death of animals following the percutaneous absorption of these substances.

Thomas Durfee, G. V. Robinson, B. B. Edwards and Arthur Kahn gave technical assistance.

Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 3, 1939.

1. Mussey, R. D.: Experiments and Observations on Cutaneous Absorption, Philadelphia M. & Phys. J. **3**:288, 1809.

2. Reilly, T. F.: The Unbroken Skin as an Absorbing Medium, J. A. M. A. **36**:250 (Jan. 26) 1901.

3. Bürgi, E.: Ueber Möglichkeiten und Grenzen einer perkutanen Therapie, Schweiz. med. Wchnschr. **67**:433 (May 15) 1937.

4. Zondek, B.: Cutaneous Application of Follicular Hormone, Lancet **1**:1107 (May 14) 1938.

5. Macht, D. I.: The Absorption of Drugs and Poisons Through the Skin and Mucous Membranes, J. A. M. A. **110**:409 (Feb. 5) 1938.

Others have reported poisonings and deaths resulting from the absorption of salicylic acid [6] and phenol [7] through the skin. Since phenol is often used in "skin peeling" preparations, caution should be exercised in its use. "Bleach creams" and "freckle removers" which contain mercury salts are also dangerous because of the possible absorption of this metal.

Various investigators [8] have reported the cure of rickets in animals and human beings by the cutaneous application of cod liver oil or ointments containing irradiated cholesterol. The cure is due to the absorption of vitamin D. Vitamin C [9] is also percutaneously absorbed, as evidenced by the increased amount in human milk after the application of an ascorbic acid solution to the intact skin of the mammae.

To the skin of spayed rats Baer [10] applied estrogenic substances contained in hydrous wool fat, petrolatum and olive oil, respectively. He observed that there was no appreciable difference in the time required for estrus to develop, irrespective of the vehicle used. He further concluded that the follicular hormone is *not* stored in the skin, since a minimal daily dose was necessary in order to maintain estrus.

MacBryde [11] treated women suffering from hypogonadism and lack of mammary development with injections of 5,000 international units per gram of estradiol or estradiol benzoate. According to the records of breast growth, a threshold was reached at which growth ceased. When injections were discontinued, a rapid regression in the size of the breast was observed. When the breasts had returned to approximately their original size, a daily unilateral application was made of 5 Gm. of the estrogenic ointment. The other breast was anointed with the same quantity of the vehicle only. The latter revealed only a slight enlargement, while the breast treated with estrogen showed a decided increase in size, and progressive growth was maintained during treatment. There were also evidences of an active estrous state, enlargement of the uterus and relief of hypogonadal symptoms. The results from a comparative study of these two forms of therapy were decidedly in favor of inunction,

6. Sannicandro, G.: Morte inopinata da applicazione di pomata salicilica (studio clinico ed anatomo-patologico e contributo alla conoscenza della cosidetta "acidosi salicilica"), Dermosifilografo **12**:273 (July) 1937.

7. Gottlieb, J., and Storey, E.: Death Due to Phenol Absorption Through Unbroken Skin, J. Maine M. A. **27**:161 (Aug.) 1936.

8. Amrhein, F. J.: Absorption of Vitamin D from the Skin, J. Am. Pharm. A. **23**:182 (March) 1934.

9. Kasahara, M., and Kawashima, K.: Die Resorption des Vitamin C durch die Haut, Klin. Wchnschr. **16**:135 (Jan. 23) 1937.

10. Baer, H. L.: The Influence of Estrogenic Substances on the Skin, J. Invest. Dermat. **2**:15 (Feb.) 1939.

11. MacBryde, C. M.: Production of Breast Growth in the Human Female by Local Application of Estrogenic Ointment, J. A. M. A. **112**:1045 (March 18) 1939.

since MacBryde observed diminishing effectiveness in breast growth when the estrogenic substance was injected, whereas when it was cutaneously applied there was a steady progressive increase in size.

METHODS FOR DETERMINING FAT PENETRATION

Since no definite technic has been evolved for the determination of fat penetration, a preliminary study was made of several methods in order to determine the best procedure to be adopted for subsequent experiments.

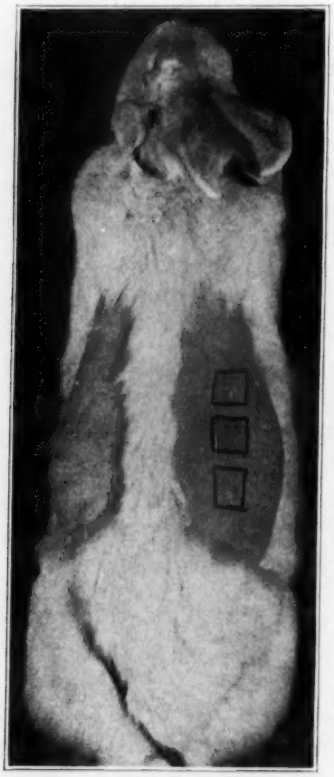

Fig. 1.—Preparation of rabbit showing areas where biopsy specimens were taken (right side). Corresponding areas of normal untreated skin were taken from opposite side as controls.

Certain factors were uniform in all methods used and will therefore be discussed here to avoid repetition.

Mature albino rabbits were used, the hair being removed from both control and treated areas with electric clippers. The materials were applied to the skin by daubing with a pad of saturated cotton, light finger massage or spraying. The more viscous products were applied in their normal state and also after being liquefied by gentle heating.

A preliminary series of biopsy specimens were taken one, two, three, four, five, six, seven, eight, sixteen and twenty-four hours after application of the products, and normal specimens were always taken from each animal, for comparison. With the materials used the rate of penetration or absorption was found to be greater between two and six hours. Therefore, these periods were adopted for the subsequent tests.

Before the biopsy specimens were taken, the areas were swabbed with 70 per cent alcohol and immediately patted dry with cotton. The procedure eliminated an excess of fat on the surface of the skin, thus permitting a more accurate reading of the slides. Sudan III and IV were used for the staining of the fat tissue. However, after preliminary studies sudan IV was adopted as the more clearly defined stain.

The various technics used for the determination of fat penetration and a discussion and criticism of each one are given herewith.

METHOD 1.—The biopsy specimen is fixed for twenty-four hours in solution of formaldehyde U. S. P. diluted 1 : 10, and sections are cut 10 microns thick with the freezing microtome. The sections are: (1) washed in distilled water, (2) put in 70 per cent alcohol for two minutes, (3) left in sudan IV for ten to fifteen minutes (covered to prevent evaporation), (4) floated onto slides, (5) rinsed with 70 per cent alcohol and then with distilled water, (6) counterstained with Harris' hematoxylin one-half minute, (7) rinsed off quickly with distilled water, (8) washed with saturated lithium carbonate, (9) rinsed with distilled water (excess water around the section is blotted up) and (10) mounted in glycerin, with the edges of the cover slip sealed with gold size.

To avoid carrying fat in from the epidermis, the biopsy specimen was reversed so that sections were cut from the subcutaneous layer toward the epidermis. The further precaution was taken of wiping the knife blade carefully before each cut. It was found that any particles of fat dragged in by means of the knife blade or by cutting from the epidermis appeared microscopically as artefacts.

METHOD 2.—The treated area is cleansed with 70 per cent alcohol and patted dry with cotton. Sudan IV, 0.05 to 0.1 cc., is injected with a 24, 25 or 26 gage hypodermic needle (½ to 1 inch [1 to 2.5 cm.] long). The biopsy cut is made just beyond the point of injection at the place where the wheal appeared, and the specimen is placed for twenty-four hours in solution of formaldehyde U. S. P. diluted 1 : 10. The sections are: (1) cut 10 microns thick with the freezing microtome, (2) washed with distilled water, (3) floated onto slides, (4) counterstained with Harris' hematoxylin for one-half minute, (5) rinsed with distilled water, (6) washed with saturated lithium carbonate, (7) rinsed with distilled water (excess water is blotted up) and (8) mounted in glycerin, with the edges of the cover slip sealed with gold size.

In the first studies this method appeared to have definite possibilities, but as subsequent experiments were carried out, its use appeared to be limited in that varying amounts of fat were found in the tissue. Intradermal injections in rabbits proved difficult, since the natural texture of the rabbit skin is relatively tough. Variations in the angle and depth of inserting the needle as well as in the time taken to expel the dye were tried in an effort to standardize the technic.

Biopsy specimens were taken at various intervals up to ten minutes after the injection of the dye.

METHOD 3.—Technic 2 was repeated with substitution of a 1 per cent solution of osmic acid for sudan IV.

The same criticism encountered with the use of sudan is applicable here, plus the fact that osmic acid is a selective rather than a total fat stain. "Osmium tetraoxide (osmic acid) is reduced to black metallic osmium by oleic acid, olein, mixed fats containing oleic acid, ethereal oil, resins, lecithin and tannins. These substances are, in turn, blackened or browned by precipitated osmium. The saturated fats and fatty acids do not reduce osmic acid." [12] A question might be raised as to why sudan III and IV are not also selective fat stains. This can be explained by the chemical structure and physical nature of the dyes. In both these dyes there is a hydroxyl group in the orthoposition on the ring in relation to the azo group. In such compounds a rearrangement of atoms to the quinoid form is certain to take place. A compound of this structure is neither basic nor acid and, not being able to form salts, does not act like an ordinary dye but is soluble in alcohol and more soluble in fat. When an alcoholic solution of the dye comes in contact with fats, the dye leaves the alcohol and dissolves in the fats, coloring them an orange-red.

The following are technics suggested by Dr. D. L. Satenstein.

METHOD 4 A.—The biopsy specimen is (1) fixed in 1 per cent aqueous solution of osmic acid, (2) washed in running water for twelve hours, (3) dehydrated through 70 per cent, 95 per cent and absolute alcohol, (4) cleared in carbon disulphide and (5) embedded in paraffin.

The sections are: (1) cut 7 microns thick, (2) mounted on slides and dried, (3) washed in: (*a*) two successive jars of xylene, (*b*) two successive jars of absolute alcohol, (*c*) 95 per cent alcohol, (*d*) acid alcohol (2 per cent hydrochloric acid in 70 per cent alcohol) and (*e*) running water, (4) stained with lithium carbonate and carmine for thirty minutes (3 Gm. of Grubler's carmine red I A boiled in 100 cc. of a saturated solution of lithium carbonate, cooled and filtered), (5) decolorized with acid alcohol for fifteen minutes, (6) washed in running water and then successively with 70 per cent, 95 per cent and absolute alcohol, (7) cleared with xylene, carbolxylene and two successive xylene baths and (8) mounted in balsam.

In the outline of this technic submitted to us it was suggested that the tissue be fixed for forty-eight hours in osmic acid. The tissue became badly macerated when left in the fixative for this length of time. Twenty-four hours proved to be ample.

This part of the technic was to establish the presence of oleic acid in normal skin. With certain preliminaries it was repeated.

METHOD 4 B.—Oleic acid was either rubbed or painted on the skin. After the appropriate intervals of time biopsy specimens of the areas were taken and the technic under 4 A was repeated.

This effort was to show whether oleic acid alone will penetrate through the skin.

METHOD 4 C.—Oleic acid (5 to 10 per cent) was added to the various products, and the compounds were applied to the animals' skins. Biopsies were made and treated as outlined under 4 A.

This technic was carried out to establish the rate of penetration of oleic acid when added to various vehicles, and the result was to be considered a measurement of penetration of each vehicle.

The entire technic could be criticized in that oleic acid, rather than any other fat, was the product measured.

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12. McClung, C. E.: Handbook of Microscopical Technique for Workers in Animal and Plant Tissues, ed. 2, New York, Paul B. Hoeber, Inc., 1937, p. 225.

Possibly owing to the previous fixation and maceration of the tissue or perhaps to something entirely independent, it proved difficult to produce a nuclear stain with lithium carbonate carmine. Many variations of the carmine stain were tried and finally ammonia-alum carmine was found to be the most satisfactory, although it did not have all the requisites of a good nuclear stain.

METHOD 5.—The biopsy specimen is: (1) fixed for thirty minutes in solution of formaldehyde U. S. P. diluted 1:2.5, (2) rinsed with distilled water, (3) placed in a sudan IV dye bath for thirty minutes and (4) rinsed with 70 per cent alcohol and then with distilled water.

The sections are: (1) cut 10 microns thick with the freezing microtome, (2) washed in distilled water, (3) floated onto slides, (4) counterstained with Harris' hematoxylin for one-half minute, (5) rinsed with distilled water, (6) washed with saturated solution of lithium carbonate, (7) rinsed with distilled water (excess water is blotted up) and (8) mounted in glycerin, with the edges of the cover slips sealed with gold size.

The difficulty encountered here was due to the fact that the dye solution and tissue were cold. In order for fats to act as solvents for the dye, they must be in either a fluid or a semisolid state. Harris' hematoxylin, however faint, masks some of the tinge imparted by the sudan. It was decided to omit the counterstain in future sections.

METHOD 6.—Technic 5 was repeated using the dye at 50 C. and keeping the tissue in the dye bath for one-half hour at that temperature.

RATE AND DEPTH OF PENETRATION OF VARIOUS FATS INTO THE SKIN, AS SHOWN BY METHOD 6

Six fats were chosen to be tested for their penetration at two, four and six hour intervals. These fats fall into three groups, namely, vegetable, animal and mineral. Olive oil and coco butter (vegetable), cod liver oil and hydrous wool fat (animal) and liquid petrolatum and petrolatum (mineral) were tested according to the following procedure.

Six mature albino rabbits were used in this series, one animal for each product. The back of each rabbit was clipped, and three small areas along both sides of the spine were defined. One substance was applied by means of a spray to each of the three areas on the left side of the animal. A 3 inch (7.5 cm.) metal collar was placed around the animal's neck to prevent licking, and the animal was kept in a stock to prevent rubbing against the sides of the cage.

Two hours after the fat had been applied the area of one of the three treated squares was gently cleansed with 70 per cent alcohol and quickly patted dry. With the use of forceps and a razor a biopsy specimen of normal untreated tissue was taken on the right side directly opposite the test area. The tissue was placed in solution of formaldehyde U. S. P. diluted 1:10 and left for twenty-four hours for fixation.

After four hours the procedure was reported using the second of the treated blocks, and, similarly, after six hours biopsy specimens of the third areas were taken.

After complete fixation the tissue was rinsed with distilled water and placed in a sudan IV dye bath that had been preheated to 50 C. and was maintained at that temperature for thirty minutes. The specimen was removed from the dye, dipped rapidly into 70 per cent alcohol and then placed in distilled water to await

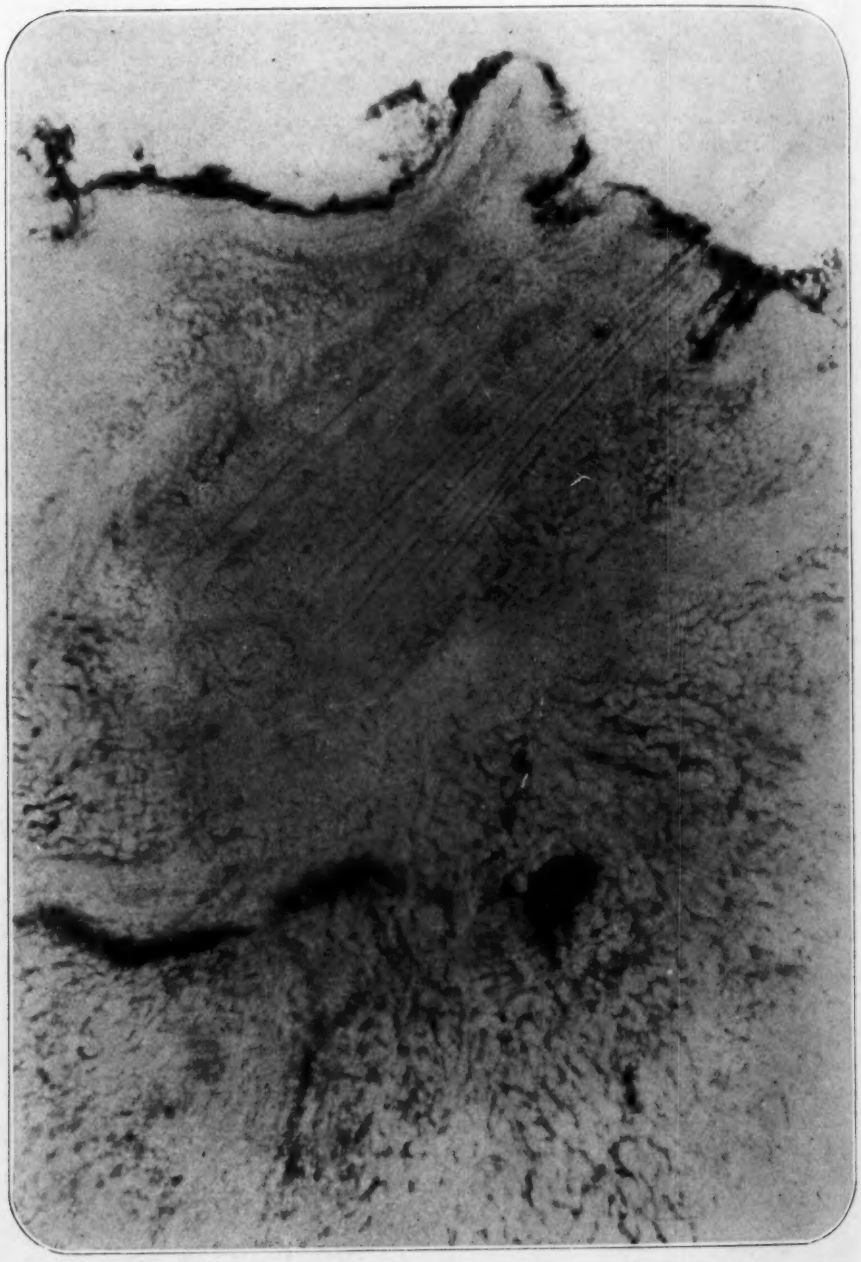

Fig. 2.—Rabbit 8, control.

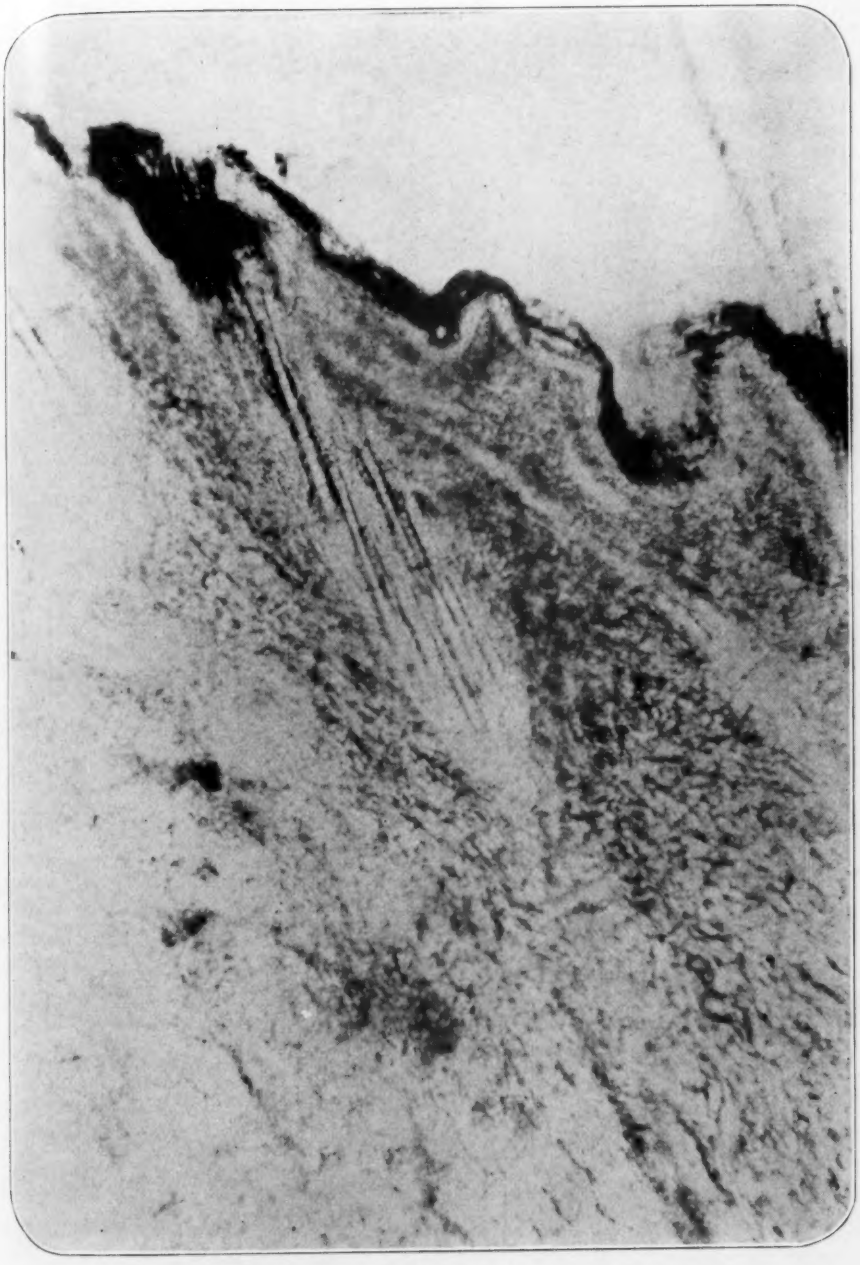

Fig. 3.—Rabbit 8, two hours after application of olive oil.

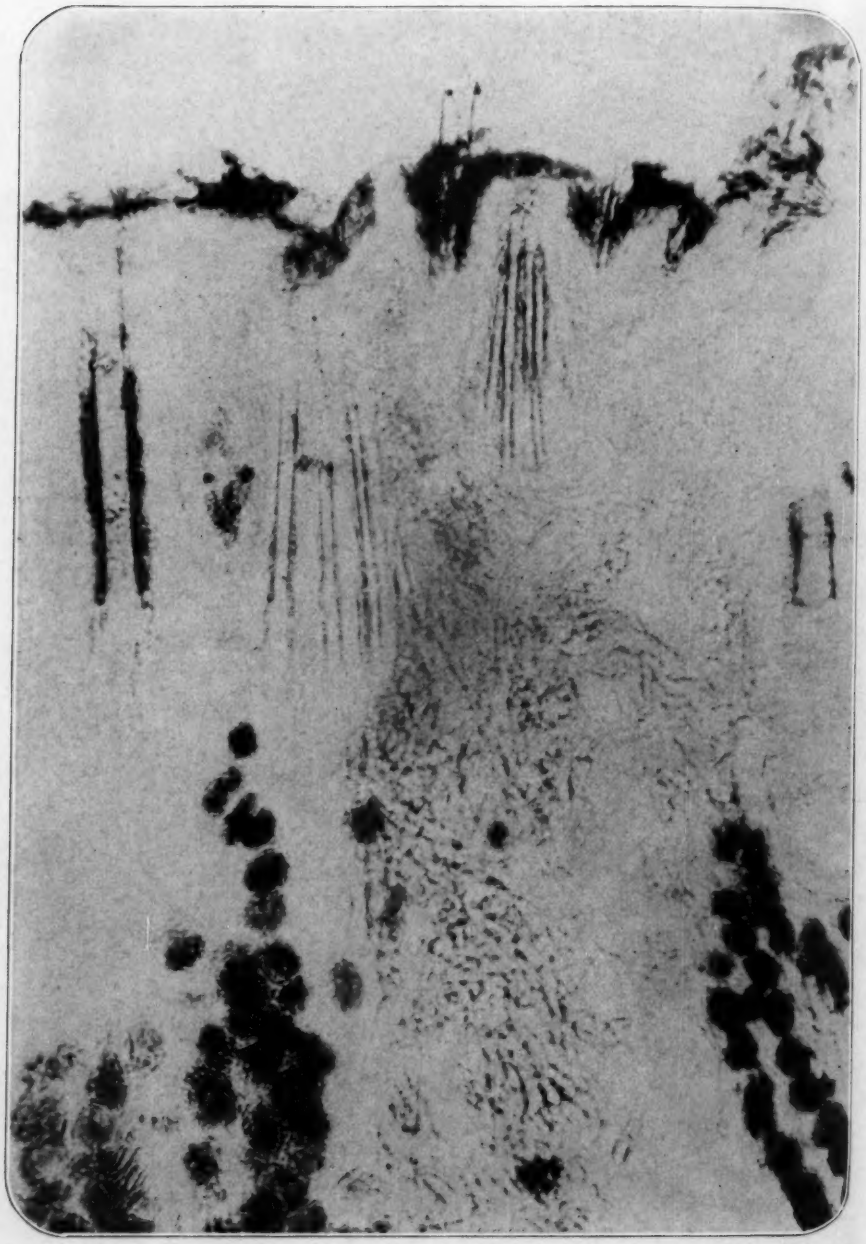

Fig. 4.—Rabbit 8, four hours after application of olive oil.

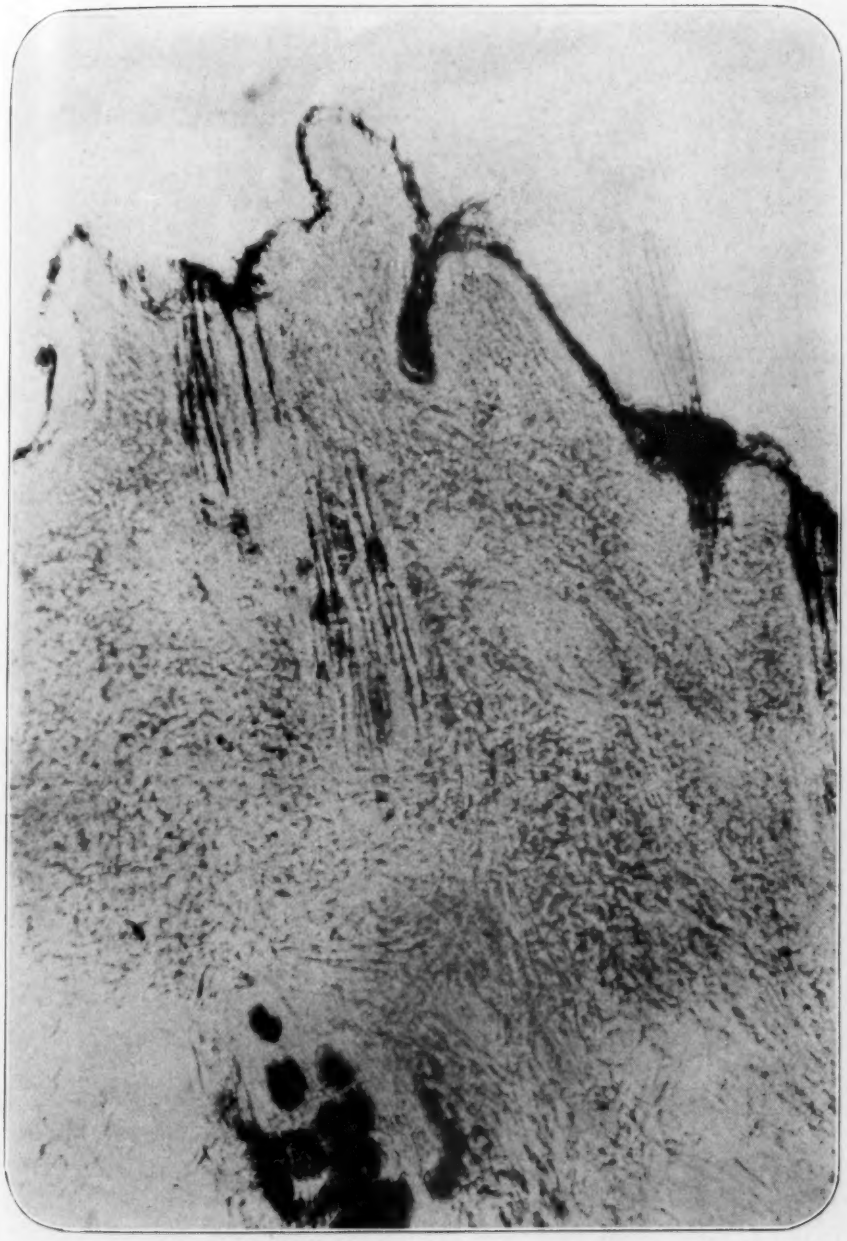

Fig. 5.—Rabbit 8, six hours after application of olive oil.

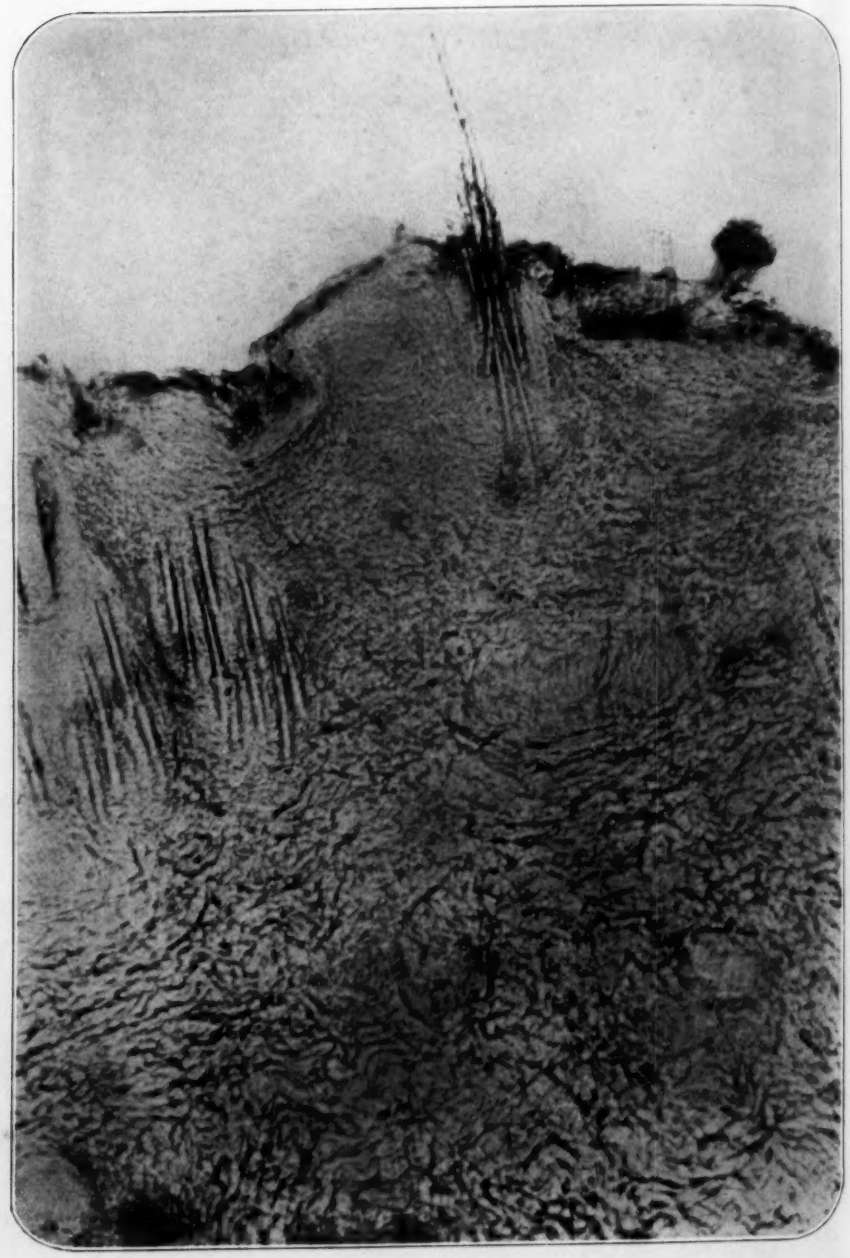

Fig. 6.—Rabbit 9, control.

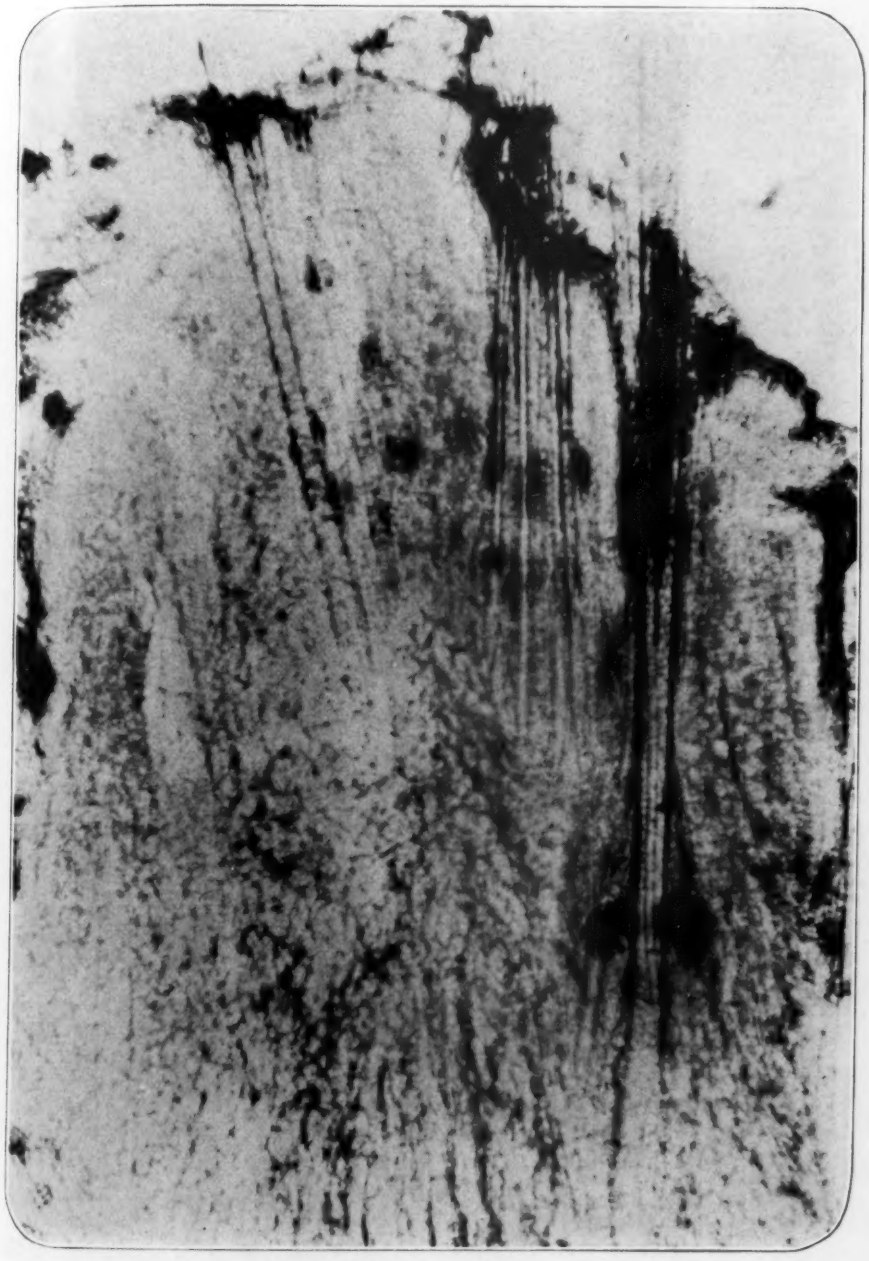

Fig. 7.—Rabbit 9, four hours after application of cod liver oil.

Fig. 8.—Rabbit 9, six hours after application of cod liver oil.

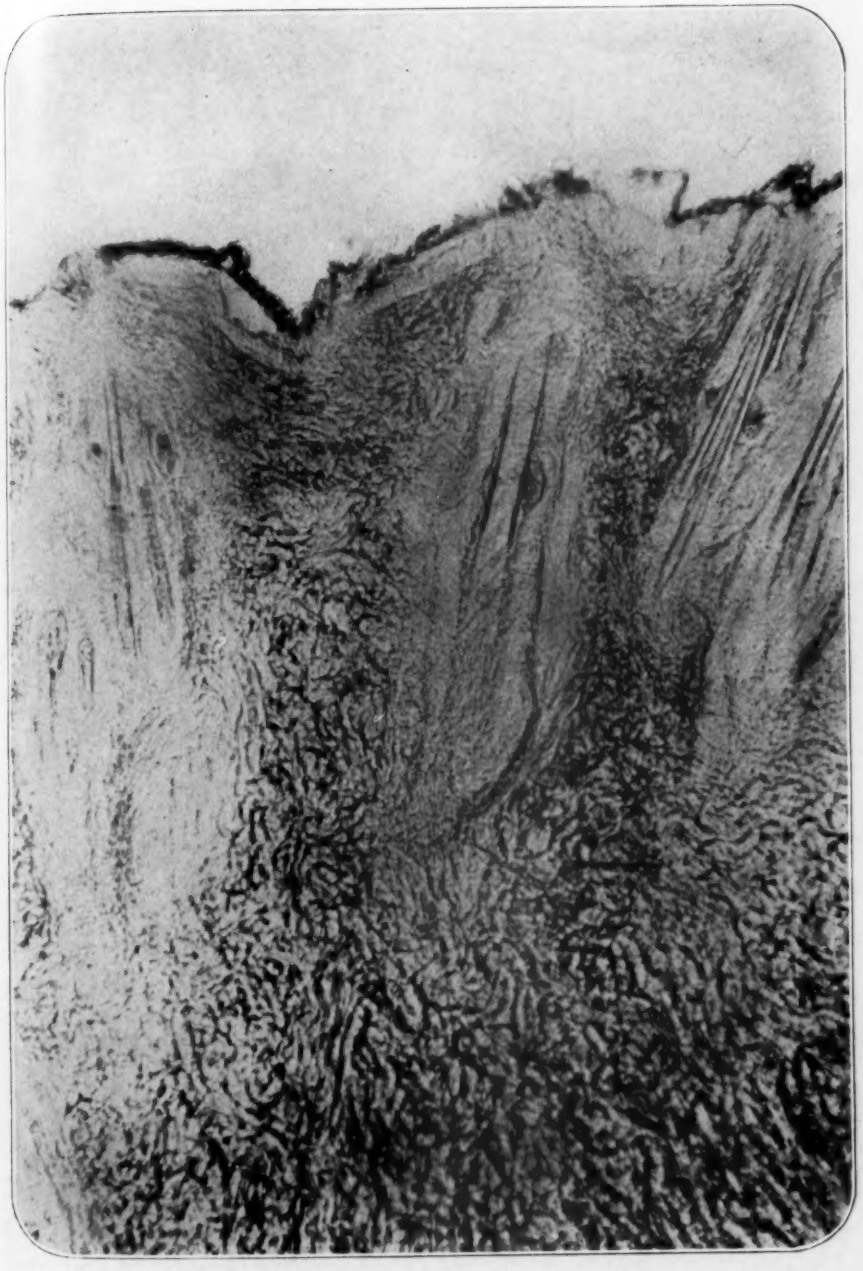

Fig. 9.—Rabbit 12, control.

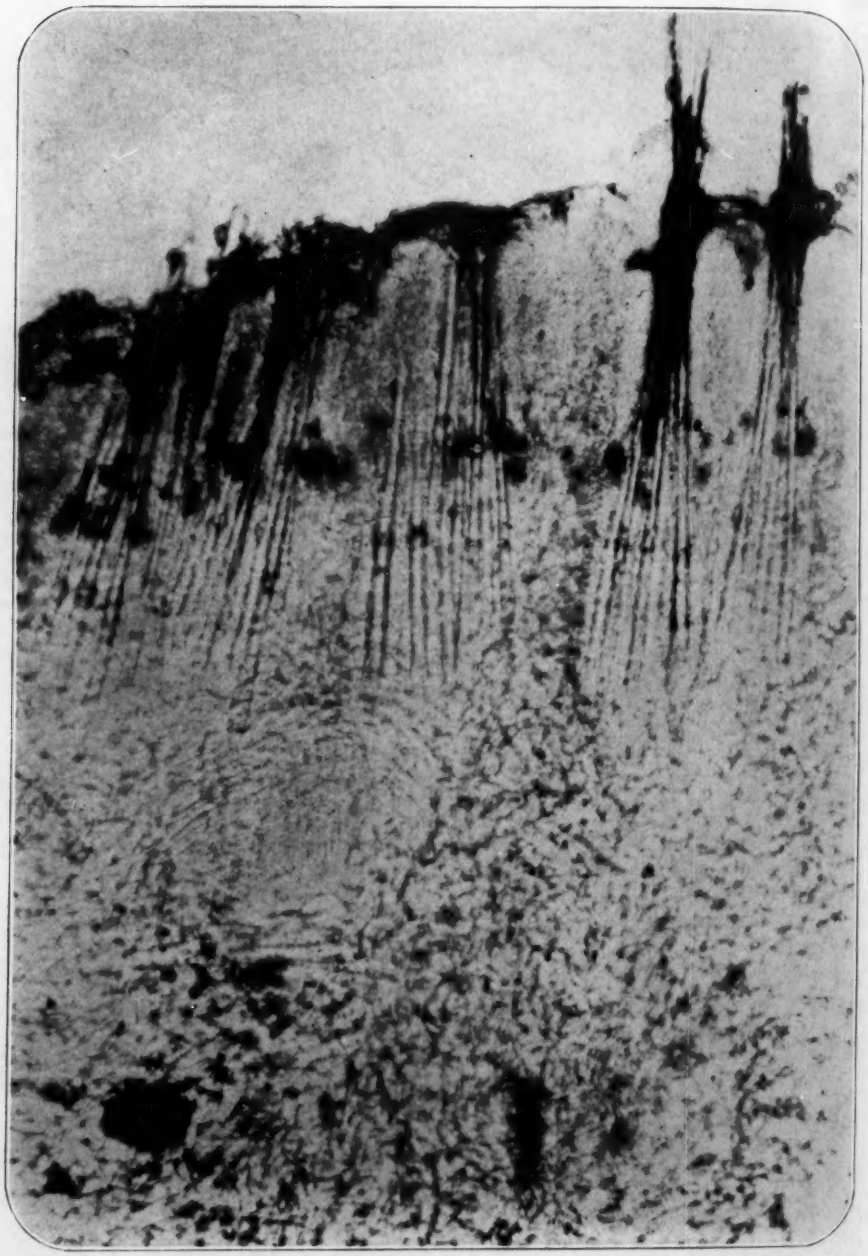

Fig. 10.—Rabbit 12, four hours after application of hydrous wool fat.

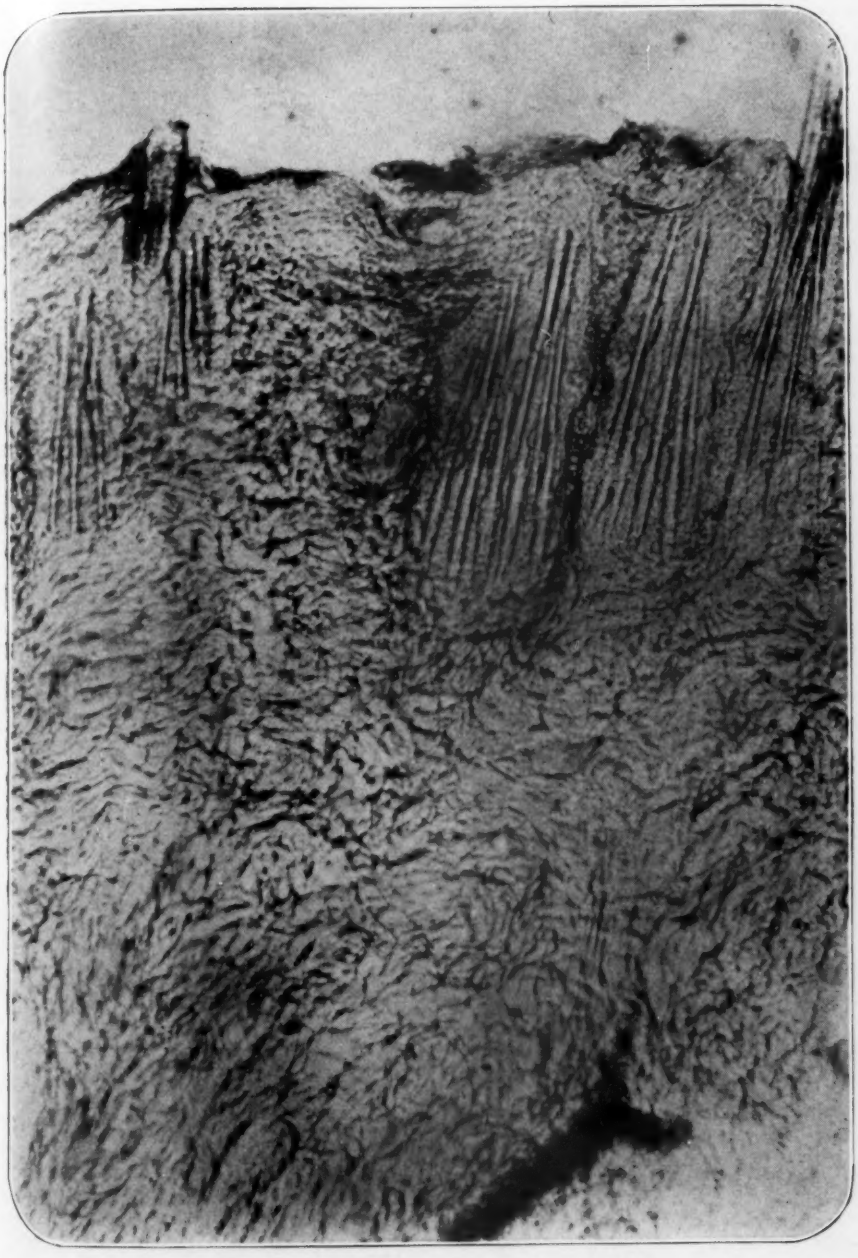

Fig. 11.—Rabbit 13, control.

Fig. 12.—Rabbit 13, two hours after application of petrolatum.

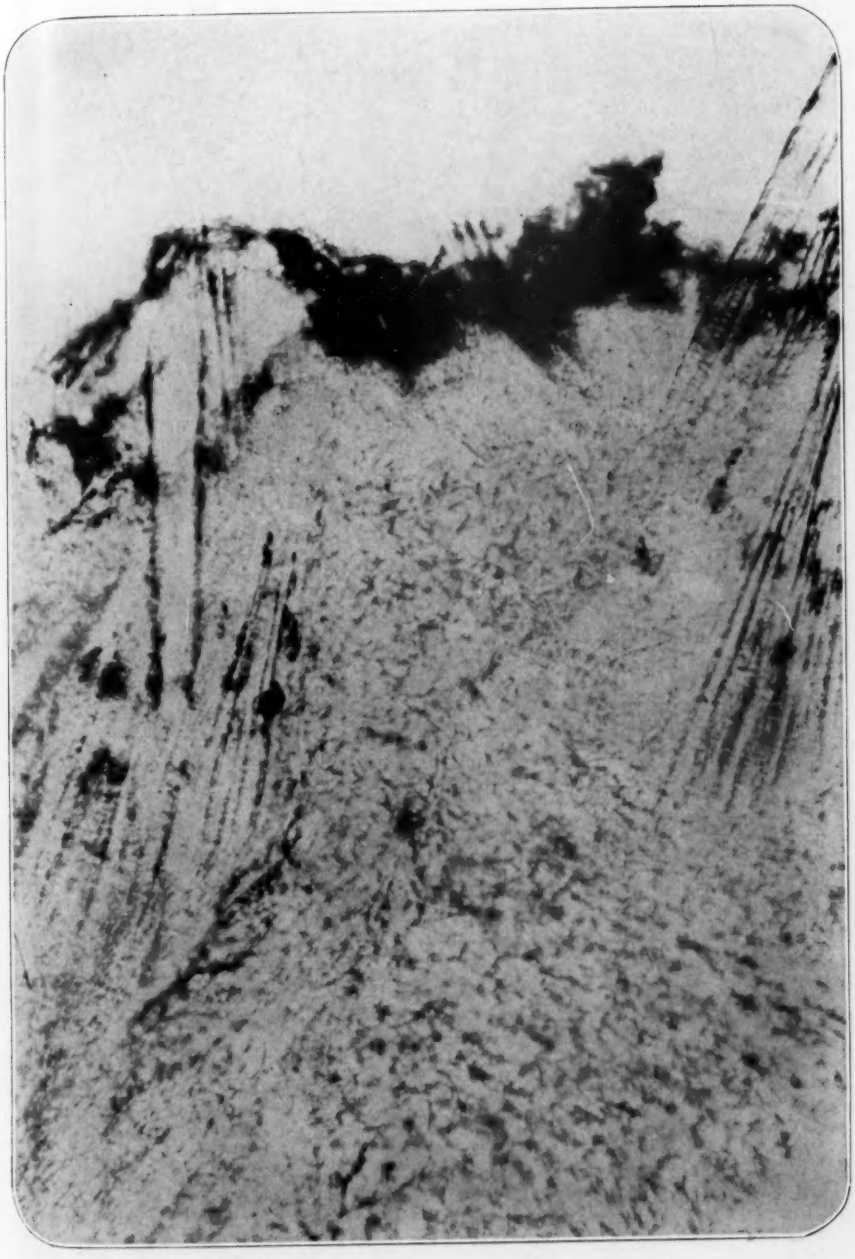

Fig. 13.—Rabbit 13, four hours after application of petrolatum.

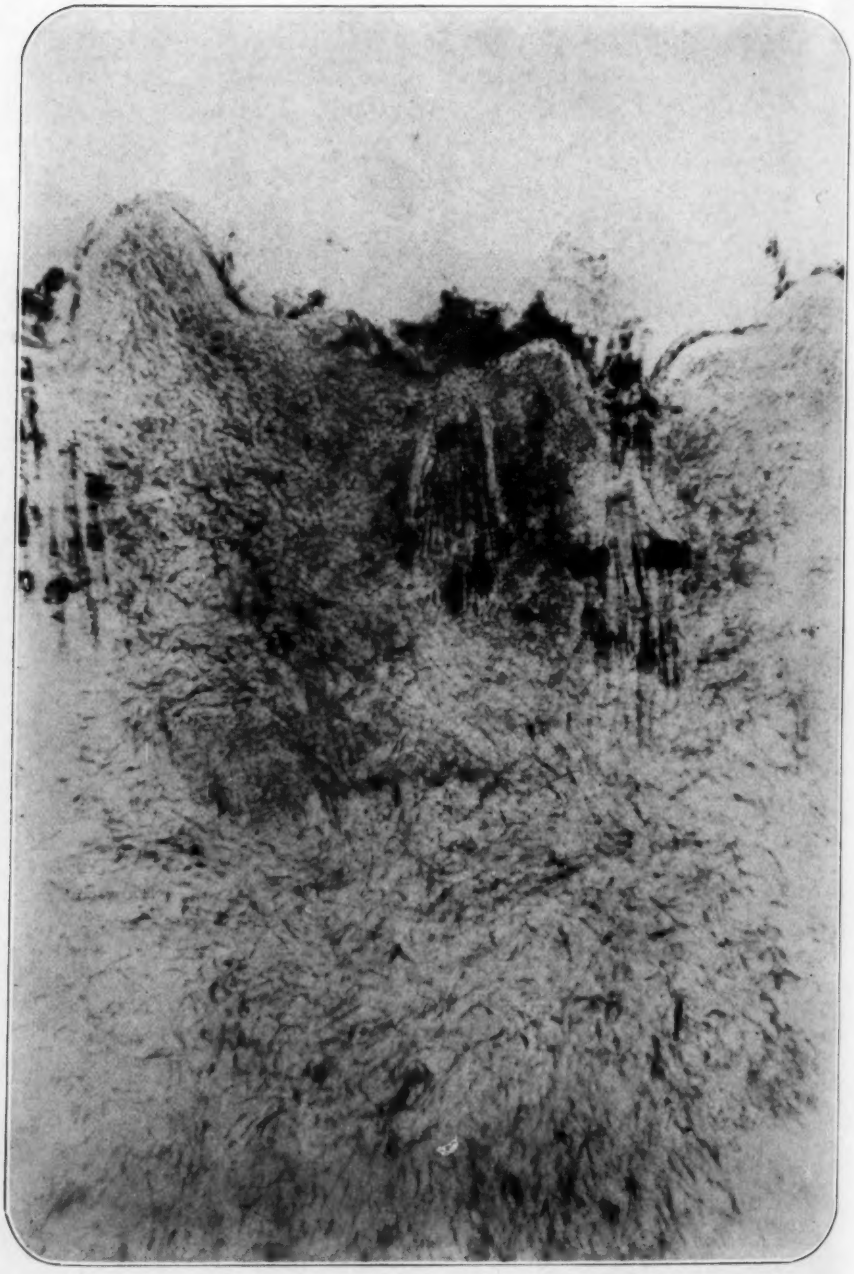

Fig. 14.—Rabbit 13, six hours after application of petrolatum.

sectioning. Sections were cut 10 microns thick with the freezing microtome. The tissue was arranged so that the knife blade passed from the subcutaneous toward the dermal layer. This order rather than the reverse was used to prevent the possible dragging of fat from the surface into the tissue as the knife passed through. The blade was cleansed after each slice was made. The sections were floated on distilled water, and four representative sections of each biopsy were mounted in glycerin and sealed with gold size. An additional fact of interest in this connection was found in those cases in which excess fat was intentionally left on the surface. In such instances, even if some of this fat was dragged into the tissue, it could be microscopically detected, since its appearance was distinctly different in contrast with the fat that had penetrated by natural absorption.

As a further precaution, it was decided to adopt the method followed in which the sections were stained in toto. These additional precautions together with those used in the earlier methods, in our opinion, obviate any possible chance of mechanical displacement of the fat.

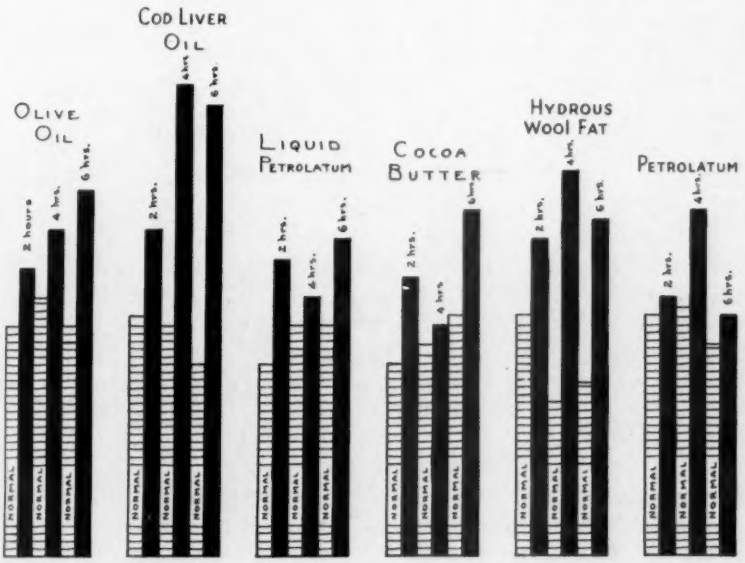

Fig. 15.—Diagrammatic comparison of penetration of animal, mineral and vegetable fats at two, four and six hour intervals (one animal per product). Note difference in fat content in untreated normal tissue taken from different rabbits for controls.

Penetration was judged by the amount of fat seen along the hair follicle and within the sebaceous glands. Definite variation was noted in the amount of fat normally found in various places along the rabbits' backs as well as in individual animals. Preliminary experiments on bilateral biopsy specimens similarly treated have shown uniform results; consequently an appropriate control specimen must always be considered with each test area.

CONCLUSIONS

1. It has already been established that:

A. Medicaments applied to the unbroken skin may be absorbed into the blood stream.

B. The rate of absorption may be influenced by the vehicle as well as by the drug it contains.

C. Volatile substances such as alcohol, ether and benzine are vehicles with a much higher rate of absorption than fats.

2. From our experiments it appears that:

A. Fats permeate the skin and do so in a large measure along the hair shafts and into the oil gland ducts.

B. Liquid fats permeate the skin more rapidly than solid fats.

C. Animal fats show the greatest dephth of penetration, with vegetable fats next and mineral fats least.

D. Most of the fats show optimum penetration between four and six hours after application. After six hours, the quantity of fat in the deeper tissues appears to diminish.

ABSTRACT OF DISCUSSION

DR. ROBERT L. GILMAN, Philadelphia: I should like to ask Dr. Eller whether he consisdered the use and study of oil emulsions on the skin, such as a mixture of hydrous wool fat, petrolatum and water or a vehicle such as lime liniment. I realize that Dr. Eller and his collaborator, Shirley Wolff, were limited in this study, but one's interest is naturally directed toward such mixtures and the effect of water in the skin-absorbing mechanism. I should like to ask Dr. Eller if the rabbit's skin was prepared by shaving, as I have in mind the rather widened absorbing area of a skin thus traumatized. The experimental animal work described will naturally lead to the use of volunteer human subjects, from whom more valid conclusions may be obtained.

DR. PAUL E. BECHET, New York: The statement that medicinal application to the skin of iodine or phenol has at times proved exceedingly dangerous and even fatal surprises me greatly. During the three years when I was an office associate of Dr. L. Duncan Bulkley, I saw him apply a 95 per cent solution of phenol to bald patches of alopecia areata up to 8 cm. in diameter. He never neutralized the phenol with alcohol. I have used the same technic for twenty-nine years in spots up to 4 or 5 cm. in diameter, and I have never observed an untoward result, either in Dr. Bulkley's practice or in my own, despite the fact that in many cases patches were repainted with phenol time and time again. Iodine is a great favorite in the treatment of dermatophytosis, and I have observed laymen, on their own responsibility, paint the entire foot with iodine without ill effect other than an occasional severe dermatitis. I do not therefore agree with the opinion that comparatively small amounts of iodine or phenol on the skin can give rise to serious or even fatal consequences.

DR. MARION B. SULZBERGER, New York: These experiments call attention to a problem which has long been recognized as important, not only in dermatology but in general biology and medicine as well. The skin is an extraordinary organ. New properties and new functions of the skin are constantly being discovered,

and observations of recent years have shown that the human skin possesses selective permeability for many categories of substances. In regard to the present experiments, it is true that results obtained with rabbits do not necessarily apply to human skin as well. Moreover, one must bear in mind that shaving, depilation or even clipping or plucking may have diminished the normal impermeability of the skin. There is, however, sufficient previous experience and evidence that these results with rabbits are applicable in a great measure to human beings. For example, it has been demonstrated that mercury penetrates the normal human skin. Although it is known that on inunction some mercury from mercury ointment is inhaled, nevertheless it has been shown that mercury does get through the human skin readily. In fact, it is now apparent that many things penetrate the skin, including simple chemicals and fats and more complex and larger molecules, such as those of certain allergens, vitamins and hormones. If, for example, vitamin D did not penetrate the skin, there would probably be a much higher incidence of rickets than there is at present; for the ray which activates the sterols to vitamin D cannot penetrate much beyond the horny layer or prickle cell layer but is filtered out at that depth. It must be, therefore, that this ray activates the lipoids at the surface of the skin and that these lipoids are subsequently absorbed. It has been proved that when these surface fats and lipoids are removed before irradiation, rickets develops unless a much larger than normal supply of vitamin D is given internally. That certain "male and female sex hormones" penetrate the skin and even produce local effects at the site of application as well as decided systemic effects has been demonstrated for animals, particularly by B. Zondek and by W. Jadassohn and co-workers (*J. Invest. Dermat.* **1**:31, 1938), and for human beings by Zondek, Zitzke, McBride and others.

I believe, however, that in adult human beings there may be a tremendous individual variation in the permeability of the epidermis to various substances. While, as Dr. Bechet said, one may use phenolic substances with impunity in most cases, there are still a few cases of fatal poisoning from phenol, even when small amounts were applied to small areas. I, myself, have seen a child die from the topical application of a lotion containing 3 per cent resorcinol. Fortunately, most skins are not as permeable as this, so that one can treat local conditions with compounds of mercury, resorcinol, tars, phenols, etc., without producing systemic effects. In treating cutaneous diseases it is to be remembered that the first few superficial millimeters only is to be treated; and I believe that in most adult skins topical remedies do not penetrate rapidly beyond this depth. One sees from the exceptional cases of poisonings what would really happen if these substances went through rapidly and in great amounts. It should be recalled that some substances, for example, alkalis, increase permeability. Washing the skin with soap may be conducive to irritation or allergic reactions by enhancing the penetration of various irritants and allergens. Substances which produce urticarial reactions usually go through the skin in certain exceptional cases; that is, only in exceptional cases in adults do the allergens which produce whealing come from without and get to the blood vessels by means of what I have alluded to as "transepidermal penetration." I believe that transepidermal penetration is more common in infants, in children and in skin which has been damaged, particularly by alkali. At the clinical presentations yesterday, a beautiful illustration of transepidermal penetration of silk allergen was presented. In Dr. Burgess' case of the young girl with atopic dermatitis, it was observed that application of an alkaline solution of silk allergen produced a wheal, even on application to unbroken skin. It seems to me that the skin is highly variable in its permeability and often very selective and that all these phenomena require much more intensive and systematic study.

DR. EUGENE F. TRAUB, New York: This paper has focused attention again on an important subject, some features of which are still highly controversial. One of the points that I believe was not stressed sufficiently at any time is the difference between the permeability of the skin to certain chemicals which allows them to be taken up in the blood stream and the absorptivity of the skin itself as an organ. I believe that more than sufficient proof has now been adduced for every one to accept the fact that certain chemicals may be absorbed through the skin and be taken up by the blood stream either for the benefit or for the harm of the patient. The second point, however, namely, the absorptivity of the skin itself or the use by the skin of an agent applied directly to the surface of the skin, presents a problem which is far from settled. Because the cosmetic business has become one of the major industries in recent years and because the value of practically all their preparations is dependent on this second factor, I had hoped that some light would be thrown on this subject. Cosmetics applied to the surface of the skin are not usually used for their general effect but for the utilization of the skin itself. They are applied, for example, as an antiwrinkle or antifreckle remedy. I wonder if Dr. Eller and his collaborator have been able to prove that any substance externally applied is absorbed solely for the benefit of the skin itself.

However, whether there is any additional information on this second point or not, any added proof that medicaments may be absorbed through the unbroken skin is valuable, because from a medicolegal standpoint, in New York at least, it has been difficult to prove in court that applications to an unbroken skin may poison some persons. In spite of considerable evidence introduced, it was difficult to prove that a suspension of 33.3 per cent mild mercurous chloride in water, rubbed on the skin over a period of twenty years, could produce a mercury poisoning simply because the mercurous chloride was applied in an aqueous suspension rather than having been incorporated in an ointment base. I wonder if Dr. Eller has worked only with preparations in a grease or an oil base or whether he has had any experiences about absorption from an aqueous vehicle.

DR. HAROLD N. COLE, Cleveland: The work presented here corroborates investigation that was done a long time ago. I certainly would take exception to the statement that no absorption takes place unless the horny layer has been damaged. It has been shown time and time again that mercury rubbed into the unbroken skin of the rat gets into the hair follicles and, combined with the chlorides in the sweat, is transformed and absorbed.

DR. EARL D. OSBORNE, Buffalo: In regard to the statement made that phenol could be used over a large surface without causing any trouble, I believe that is not entirely true. Deep sloughing following local applications of very weak phenol preparations has been reported, as well as collapse from painting on fairly concentrated solutions for local effects. I have seen a large area of the skin and subcutaneous tissue slough from the chest after the application of an ointment containing 0.5 per cent phenol. In another case, that of a young girl with a flat pigmented nevus, an area 5 cm. in diameter was painted with concentrated pure phenol. Within a few minutes she suffered a circulatory collapse, and the respiration became extremely shallow. Fortunately, she recovered. I believe that phenol is extremely valuable in many cases, but it can and does produce serious effects in some persons.

DR. E. WILLIAM ABRAMOWITZ, New York: The absorption of fats by the skin is dependent among other things on their melting points. Burgi has demonstrated that pure mercury in the form of gray mercury ointment penetrated the skin more easily than ammoniated mercury. Mercury bichloride in a 1:1,000 solution

penetrated little, and in a 1:100 solution not at all (Current Comment, J. A. M. A. **107**:1722 [Nov. 21] 1936). It is known that boric acid solution used as a foot bath is capable of changing the reaction of the urine to the acid side, indicating the absorption of boric acid through the skin.

Dr. Paul E. Bechet, New York: Perhaps I may be allowed a few words in rebuttal of Dr. Osborne's discussion. I should be the last one to advocate applications of a 95 per cent solution of phenol over large areas, but I have treated areas of alopecia areata, even up to 4 or 5 cm. in diameter, hundreds of times and have never observed the slighest deleterious effect. I wonder if Dr. Osborne's patient did not collapse through hysteria or autosuggestion, much as soldiers occasionally faint while waiting in line for a hypodermic injection.

Dr. Joseph J. Eller, New York: In answer to Dr. Gilman's question, experiments have shown that any drug, chemical or other material soluble in oil or water can permeate the skin. In our experiments the hair on the animals was cut; shaving was avoided to prevent trauma. Regarding Dr. Bechet's discussion of phenol, I think that this can be explained. What Dr. Bechet and most therapeutists do for alopecia areata is to paint on a solution of almost pure phenol which is not absorbed. A weaker solution will go through the skin, but a 95 per cent solution precipitates on the surface, with minimal absorption. There are, however, several reports of instances in which a solution of phenol applied to a small area on the skin of a child resulted in death. Dr. Traub's comments are interesting. When certain substances are applied to the skin there is penetrability. The materials either go along the hair shafts into the cutis and remain there and are excreted onto the surface of the skin again or are absorbed in some way into the blood stream, with the possibility of producing systemic effects. Regarding the local effects of substances applied to the skin, I do not know of any experiments that have proved that a substance applied locally has increased the local metabolism or caused rejuvenation of the tissues or cells locally.

PYODERMA (ECTHYMA) GANGRAENOSUM

DOUGLASS W. MONTGOMERY, M.D.
SAN FRANCISCO

There occurs rarely on the skin a lesion resembling an ulcer that spreads slowly and obstinately with a gangrenous border. It often is connected with, and evidently arises from, a preceding suppurative lesion and usually occurs in a person afflicted with some debilitating disease, but in the case reported here it was not so associated. On the contrary, neither ulcerative colitis nor other suppurative disease was present. The patient, it is true, had been under a heavy strain for some months, attending an ailing sister and holding her position as bookkeeper, entailing both excess of work and loss of sleep.

This gangrenous process, attributed, as in the present case, to a streptococcus, is now more than usually interesting because of the remarkable influence of sulfanilamide on streptococcic diseases. I report a failure of sulfanilamide after two trials in this case.

The disease has a bewildering number of names, and I have chosen pyoderma (ecthyma) gangraenosum, as given by Brunsting, Goeckerman and O'Leary.[1] The insertion of the word ecthyma I regard as being particularly appropriate, indicating a relation to a similar variety of streptococcic disease.

REPORT OF A CASE

A vigorous energetic woman of 57 years consulted me Sept. 9, 1938, for a lesion that resembled an ulcer situated on the outer surface of the lower third of the left leg. The surface of the lesion was covered with what seemed to be bright red granulations, and the borders were raised, giving the base the appearance of being depressed. The upper advancing border differed from the rest in that it was occupied by a dirty slate-colored moon-shaped necrotic flap, fully half an inch in breadth. Beyond this the skin was smooth, slightly swollen and bluish red.

The lesion was first noticed six weeks previously as a red area shining through her stocking. Her physician was called, and a small quantity of pus was evacuated, but the lesion refused to heal. Cultures were made, and at first only Staphylococcus aureus was found, but later streptococci were also demonstrated.

Shortly after I first saw the patient, there appeared a circular, bright red disk on the outer surface of the lower third of the right leg, an intertrigo in the right groin and a rash over the buttocks. There was no doubt about these lesions being

1. Brunsting, L. A.; Goeckerman, W. H., and O'Leary, P. A.: Pyodermia (Ecthyma) Gangrenosum: Clinical and Experimental Observations in Five Cases Occurring in Adults, Arch. Dermat. & Syph. **22**:655 (Oct.) 1930.

streptococcic, and they cleared up under the usual treatment. These of course contributed to the impression that the main lesion was also streptococcic, although in some respects it acted very differently.

The process began the last days of August 1938, and the advance ceased the last of February 1939. During the intervening six months it traveled slowly and steadily from the ankle to above the knee, a distance of 13 inches (33 cm.). The advance was almost continuously upward. Twice it passed a short distance across the crest of the tibia, and once it threatened to invade the popliteus. Two notable features of the raw surface were its excessive tenderness, far beyond what an ulcer ordinarily exhibits, and its quick healing, not alone from the edges but also from islets scattered over its surface.

The necrosis had a curious geometric advance. Healing of the raw surface took place, pointing up to the center of the crescent, and then the crescent parted, each portion traveling upward, and slightly outward (fig. 1). One of the portions

Fig. 1.—The upper thick border of the crescent and the lower healing border pointing up at its center, which will ultimately split the crescent in two. To the left is the scaly healed surface over which the ulcer has traveled. (Photograph by Dr. James D. Viecelli.)

died away while the other continued, and then the same process was repeated. This course persisted until the very last. Finally, while one crescent advanced up toward the knee, the other swung around over the knee, and on meeting head-on they both were extinguished, with startling suddenness. After this collision there still remained a small segment of the inner part of the upper crescent, about 1 inch (2.5 cm.) in length, which had a sinuous slender rolled border. This border was light yellow and glistening and resembled the rolled border of an epithelioma. There was, however, absolutely none of the undercutting so characteristic of the earlier stages of the disease. This in its turn faded out in a few days, leaving a surface which healed with its accustomed rapidity.

What caused this sudden stop in the disease process I, of course, do not know. It may have been a serum, though this is unlikely, or it may have been the lack of nourishment, as it was noticed that the process

did not return over its track. When the two crescents met they had no food for a forward advance, and their reluctance for a lateral one had been noticed.

The statement that the process did not return over the healed area is not quite correct. Blisters formed from time to time, some of which were of the size of blebs. This was considered quite in line with the nature of the virus, as the streptococcus is decidedly serotaxic.

Fig. 2.—The healed process. In the center, over the knee, is an area of normal skin that never was invaded. One ulcer traveled up around this to the right, and another advanced up to the left, and on meeting both were extinguished. Over the healed surface are many lines and areas showing still active hyperemia, together with scattered patches of skin which have returned to a normal appearance. (Photograph by Dr. James D. Viecelli.)

Nine months after the commencement of the disease the area traversed was still red and easily wounded, but at the borders the skin was assuming its normal color, and islands of normal skin were enlarging throughout. Whatever scar will remain will, for almost the whole surface, be very thin. Curiously enough, over the knee, where the process ceased, there will remain quite a prominent scar with clawlike projections.

Before I undertook the treatment of the patient, sulfanilamide had been administered in large doses by competent physicians, but fruitlessly. Warned by this, but still hopeful because of the success of Goodman,[2] I employed neoprontosil (disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid) locally, but soon the patient exhibited the poisonous effects of the drug with no local improvement. This breakdown of the morale of the patient was a serious matter in a disease in which the main reliance is on corporeal resistance.

The wound was exposed to sunlight every day, and the ingestion of an adequate quantity of oxygen was secured by both massage and as much voluntary exercise as possible for a patient confined to bed. She took a tablespoonful of cod liver oil three times a day. On giving the cod liver oil, of course, the milk fat was eliminated; otherwise the patient would have received an excess of fat. She also was given 0.78 Gm. (12 grains) of pills of ferrous carbonate three times a day. Whitfield, of London, is the only man in recent times who has mentioned the good effect of iron in streptococcic infections.

The best dressing for the lesion, and many were tried, was an ointment composed of 20 per cent Peruvian balsam and 30 per cent zinc oxide in equal parts of cod liver oil and wool fat.

Although the lesion always resembled a deep ulcer, the ulceration for almost the whole surface was remarkably shallow, showing that the infective agent lived almost exclusively in the mucous layer, causing the death of this layer and of all the layers above it. This also accounted for the excessive tenderness, as the nonmedullated nerve fibers running up into the mucous layer are particularly organs of itching, tickling and pain.

At no time was the lymphatic system affected, and no swelling was ever demonstrable in the glands in either the saphenous opening or the groin. The absence of glandular involvement in what appeared to be such a large formidable ulcer was most remarkable, when one reflects what insignificant suppurative lesions will affect the lymphatic system.

On observing the course of the disease, the evidences of superficiality became so clear that they checked any desire to perform an adequate biopsy, for fear of introducing the virulent agent into the deeper tissues.

The temperature of the patient was only slightly affected. This conformed with what is known of ecthyma, which this lesion so closely resembled and in which glandular enlargement, lymphangitis, phlebitis and abscesses are rarities.[3]

The most remarkable features, the rapidity of healing, with scattered islands of epithelium over the raw surface, and the healing with what appeared to be a thin scar, were noticed by the physicians in the Mayo Clinic and by several others.[1] For instance, Sabouraud, in speaking of a condition which he called fulminating gangrene, stated that it healed with incredible rapidity, without any excessive cicatricial retraction and with a remarkable conservation of the normal lines of the region. In a case which he examined bacteriologically the lesion literally swarmed

2. Goodman, M. H.: Chronic Streptococcic Ulcer of the Skin Unresponsive to Local Therapy, But Cured by Sulfanilamide: Report of Two Cases, J. A. M. A. **111**:1427 (Oct. 15) 1938.

3. Darier, J.: A Textbook of Dermatology, translated by S. Pollitzer, Philadelphia, Lea & Febiger, 1920, p. 170.

with streptococci.[4] I think that Sabouraud was dealing with the same disease that my patient suffered from. Balog [5] also found, in all his cases of chronic ulcerative pyoderma, epithelial islands in the center of the ulcerations and at the steep undermined edges and thought that these islands were from the epithelium of the deep-seated sweat glands.

There is also a circinate erosive balanoposthitis which resembles this condition very closely and which has the characteristic of quick healing.[6]

At one time in the course of the disease the patient was seized with a sharp attack of diarrhea that caused no little concern, as many patients with this disease have colitis, which increases the gravity of the condition.[7]

In one of Brunsting's patients the ulcer began as a pustule with an inflammatory areola. In my patient, as previously related, it began as a bright red spot which quickly underwent a suppurative change. It may be inferred that the red spot was similar to that which later appeared over the outer side of the right ankle, the intertrigo in the right groin and the rash over the buttocks. All these were clearly streptococcic.

Although the appearance and progress of this lesion differed decidedly from what one finds in streptodermatitis, fundamentally it differed only in degree. Ordinarily the streptococcus flourishes in the mucous layer of the skin and, being serotaxic, causes a blister under the horny layer. Rarely does it cause glandular enlargement, lymphangitis, phlebitis or abscesses. In this case for some reason the virus acted profoundly and tenaciously on the mucous layer of the skin at the upper border of the lesion, not on the other borders, and, after pursuing an uncontrollable course for six months, finally stopped on colliding with itself.

450 Sutter Street.

4. Sabouraud, R.: Manuel élémentaire de dermatologie topographique régionale, Paris, Masson & Cie, 1905, p. 484.

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CUTANEOUS MANIFESTATIONS OF PERIARTERITIS NODOSA

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Periarteritis nodosa is an inflammatory disease affecting the smaller arteries and arterioles. Almost all the organs and tissues of the body may be involved, giving rise to a variable train of symptoms, depending on where the condition has particularly established itself. The vascular changes may result in a partial or complete cutting off of the blood supply causing impairment of function or localized areas of cell death.

Alkiewicz [1] collected from the literature between 1866 and 1933 36 cases in which the skin was affected. In 16 of these there were small macules *(kleine Flecken)*, which were purpuric in 7; in 11 there were nodules; in 15, papules or small nodules; in 7, severe hemorrhages, and in 5, necrosis. In a fairly exhaustive survey of the literature from 1912 to the present we have been able to add 23 cases to those published by Alkiewicz. With over 200 reports in the literature to date, these figures would indicate that the skin is affected in about 25 per cent of cases. Nodules are most commonly recognized as the characteristic pathologic evidence of the disease. They may occur in the skin or subcutaneous tissues, are usually movable, appear in crops, are seldom painful and may reach the size of walnuts. Purpura alone or complicating other lesions is relatively frequent, and erythematous, vesicular, urticarial and scarlatiniform rashes have been met with.

Hutinel, Coste and Arnaudet [2] published a report on a girl aged 10 who had abdominal colic, articular pain, intestinal hemorrhages and a scarlet fever-like rash over the lower extremities. This rash later disappeared and was followed by a purpuric eruption, the lesions of which

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2. Hutinel, J.; Coste, F., and Arnaudet, A.: Péri-artérite noueuse de Kussmaul forme spléno-intestinale, Arch. de méd. d. enf. **33**:355 (June) 1930.

were the size of freckles, deep red, elevated and did not disappear on pressure. In some of these lesions vesicles developed containing a seropurulent or sanguineous fluid; later necrosis and ulceration developed, and the patient died. Autopsy revealed typical pathologic changes

Cases of Periarteritis Nodosa with Cutaneous Lesions Not Reported by Alkiewicz [1]

Year	Author	Age	Type of Lesion
1912	Beattie, J. M., and Douglas, A.: J. Path. & Bact. **17**: 195, 1912	19	Hemorrhagic macules on anterior surface of legs
1923	Kopp, G.: Deutsche med. Wchnschr. **49**: 1239, 1923	60	Generalized nodules
1923	Ophüls, W.: Arch. Int. Med. **32**: 870, 1923	38	Purple macules on thighs
1925	Keegan, J. J.: Arch. Int. Med. **36**: 189, 1925	24	Pigmented areas on thighs, arms and chest
1928	Debre and associates [3]	3	Small nodules, papulovesicles and bullae, lesions of the mouth, necrosis and ulceration
1929	Van Paassen, W.: Nederl. tijdschr. v. geneesk. **73**: 475, 1929	21	Generalized nodules
1930	Hutinel, Coste and Arnaudet [2]	10	Scarlet fever-like rash, later purpuric papulovesicular eruption
1930	Herlitz, G.: Acta pædiat. **10**: 105, 1930	8	Hemorrhagic blotches on hand, with gangrene of a finger
1931	Goldstein, J.: Wien. Arch. f. inn. Med. **21**: 255, 1931	17	Subcutaneous nodules
1932	Schottstaedt, W. E. R.: California & West. Med. **36**: 186, 1932	41	Subcutaneous nodules
1934	Goldschlag and von Chwalibogowski [5]	5	Livedo racemosa, polymorphous rash and macules
1934	Crosti [6]	5	Livedo racemosa, polymorphous erythema and small and large nodules
1934	Barnard, W. G., and Burbury, W. M.: J. Path. & Bact. **39**: 285, 1934	8	Purplish discoloration of the limbs, gangrene of the fingers and toes
1935	Krahulik, L.; Rosenthal, M., and Laughlin, E. H.: Am. J. M. Sc. **190**: 308, 1935	9	Purpuric and ulcerated area on ankle
1936	Carol and Prakken [7]	19	Nodules, livedo reticularis limited to the wrist
1936	Lummis, F. R.: Arch. Int. Med. **10**: 105, 1936	10	Scarlet area on calf
1936	Dunbar [9]	15	Purpuric blotches and cyanotic tint on extremities
1936	Motley, L.: J. A. M. A. **106**: 898, 1936..	31	Purpuric lesions and nodules
1936	Motley, L.: J. A. M. A. **106**: 898, 1936..	35	Red nodules in the skin
1936	Cohen, Kline and Young [10]	27	Cyanosis and gangrene of hand
1938	Ward [8]	7	Livedo reticularis and purpura on the extremities
1939	Weir, D. R.: Am. J. Path. **15**: 79, 1939..	30	Ham-colored lesions on fingers and toes; petechiae beneath fingernails and toenails and on abdomen
1939	Ketron and Bernstein	37	Livedo reticularis with necrosis and ulceration

in some of the internal organs. Histologic examination of a bullous lesion on the skin revealed a picture similar to that seen in polymorphous erythema. The arterioles and capillaries in the cutis were filled with polymorphonuclear leukocytes, and there was an infiltration of the adventitia and periadventitial tissues with the same cells. The media, however, was intact.

Debré and associates [3] published a report on a 13 year old child in whose skin hemispheric nodules developed, varying from 3 to 7 mm. in diameter, with papulovesicles or bullous lesions, containing a serosanguineous fluid, and some ecchymotic patches. This patient also presented a few small nodules and sanguineous erosions on the lips, tongue and rhinopharyngeal mucosa. The lesions on the body became necrotic and ulcerated.

Severe hemorrhages may occur, as in the case reported by Freund.[4] In this patient, a woman aged 63, they were found in the subcutaneous tissue and also in the parenchyma of the breasts. One on the leg reached the size 18 by 32 cm. Some of these lesions became gangrenous and ulcerated, but they all healed, the last one a year before the patient's death.

The first case of periarteritis nodosa with livedo reticularis or livedo racemosa was described, according to the authors themselves, by Goldschlag and von Chwalibogowski [5] in 1935. A boy aged 5 presented dark blue livedo racemosa on the extremities and trunk, particularly marked on the back. The larger strands were slightly elevated and could be palpated. There was also a polymorphous rash consisting of urticarial lesions, macules, papules and nodules. This patient was observed for about one year, during which time he had several relapses. At the end of the period of observation his skin was clear except for mild remains of the livedo. The diagnosis was established by histologic study of a nodule from the arm.

In the same year Crosti [6] published a report on a 5 year old boy who presented lesions of the skin throughout the entire seven months of his illness. There was livedo racemosa as well as polymorphous erythema, with the development later of nodules in the skin varying in size from that of a millet seed to that of a pea. Some larger nodules were also found in the deeper tissue. Here too the diagnosis was made from one of the nodules in the skin. Some months later the patient appeared to be in perfect health.

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3. Debré, R.; Leroux, R.; Lelong, M., and Gauthier-Villars, P.: La première observation française de périartérite noueuse, Arch. de méd. d. enf. **31**:325 (June) 1928.

4. Freund, F.: Apoplexia Cutanea-Periarteriitis nodosa, Arch. f. Dermat. u. Syph. **152**:158, 1926.

5. Goldschlag, F., and von Chwalibogowski, A.: Ueber einem Fall von Periarteriitis nodosa mit ausgebreiteten Hauterscheinungen, Arch. f. Dermat. u. Syph. **171**:622, 1935.

6. Crosti, A.: Contributo alla conoscenza dei quadri clinici e dermatologici della periarterite nodosa e degli eventuali loro rapporti con l'infezione reumatica, Gior. ital. di dermat. e sif. **76**:15 (Feb.) 1935; abstracted, Wise, F., and Sulzberger, M. B.: Year Book of Dermatology and Syphilology, Chicago, The Year Book Publishers, Inc., 1935.

The patient of Carol and Prakken,[7] a girl aged 19, showed livedo reticularis limited to the wrist, associated with cutaneous and subcutaneous nodules. The diagnosis again was made from a histologic study of one of these lesions.

Ward [8] described a child of 7 years who presented a striking blotchy maculopapular eruption with irregular, slightly raised borders on her face, arms, legs and, to a lesser degree, back. The lesions varied from red to light purple, and the centers were white. This patient was seen at the Harriet Lane Home of the Johns Hopkins Hospital by some of the members of the dermatologic staff who stated that the changes in the skin were of the livedo reticularis type.

Other cases have been published in which rashes of a blotchy character have been noted that very likely may have shown this pattern, although it was not specifically mentioned. Dunbar,[9] for example, in the case of a white girl aged 15, stated that there was a diffuse cyanotic tint on the extremities where numerous large and irregular purpuric blotches were conspicuous. In the patient observed by Cohen, Kline and Young [10] there was cyanosis of the left hand with beginning gangrene of the thumb and of the tips of the index finger and little finger.

The pathology of periarteritis nodosa has been exhaustively presented by Arkin,[11] who divided the arterial changes into four stages.

The first, or degenerative, stage is characterized by alternative degenerative changes in the media, with edema and the appearance of a thready fibrinous exudate about the elastica interna. There is swelling of the muscle cells, and a part of the entire circumference of the vessel wall undergoes coagulation necrosis with a hyalin-like appearance of the inner media. The endothelium may also be affected, so that the cells become desquamated or disappear over the affected area, or it may be elevated by the edema and fibrinous exudation, causing it to bulge into the lumen. Leukocytes begin to wander into the necrotic area, but in this stage the adventitia is often unaffected.

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7. Carol, W. L. L., and Prakken, J. R.: Die kutane Form der Periarteriitis nodosa, Acta dermat.-venereol. **18**:102 (Feb.) 1937; abstracted, Wise, F., and Sulzberger, M. B.: Year Book of Dermatology and Syphilology, Chicago, The Year Book Publishers, Inc., 1937.

8. Ward, R.: Periarteritis Nodosa, in Brennemann, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Company, Inc., 1937, vol. 3, chap. 13, sect. 2, p. 71.

9. Dunbar, J. C.: Periarteritis Nodosa with Associated Thrombocytopenic Purpura, Bull. School Med. Univ. Maryland **20**:138 (April) 1936.

10. Cohen, M. B.; Kline, B. S., and Young, A. M.: Clinical Diagnosis of Periarteritis Nodosa, J. A. M. A. **107**:1555 (Nov. 7) 1936.

11. Arkin, A.: A Clinical and Pathological Study of Periarteritis Nodosa: Report of Five Cases, One Histologically Healed, Am. J. Path. **6**:401 (July) 1930.

In the second, or acute inflammatory, stage a great infiltration of the media and adventitia rapidly takes place, with polymorphonuclear neutrophils and sometimes also with many eosinophils, lymphocytes and plasma cells. The perivascular connective tissue becomes edematous and the leukocytes appear in large numbers. At the height of this stage secondary thrombosis of the lumen with infarction of the various organs is common.

In the third, or granulation tissue, stage the fibrin network and hyalinized necrotic media are gradually replaced by the cellular granulation tissue, which not only replaces the destroyed media but extends outward through the adventitia. In addition to the granulation tissue formation there is usually a decided reactive proliferation of the intima, with a partial or total occlusion of the lumen. The spread of the intimal proliferation beyond the area of destruction of the media explains why we so often find in transverse sections a marked intimal thickening without changes in the vessel wall.

In the fourth, or healed granulation tissue, stage the lumens of the vessels are greatly reduced in size or totally obliterated; the destroyed arterial wall is replaced by scar tissue, and there is a decided periarterial fibrosis.

REPORT OF A CASE

History.—A. H., a white man aged 37, a structural iron worker, came to the skin clinic of the Johns Hopkins Hospital on June 12, 1937, complaining of a rash. He was placed in the medical ward, where he remained until the time of his death on August 1, a period of seven weeks.

His general health had always been good. There was no history of rheumatism. Two years previously he had had an attack of quinsy which lasted about one week. He was operated on twice in 1924 for a broken arm. In 1932 he received a traumatic injury to the right knee, which has since become occasionally swollen and sore after a strain.

The eruption appeared about ten weeks before admission, with the development of bluish streaks on his shoulders; later he noted a flushing of his face. He consulted a physician who gave him ultraviolet radiation and three injections in the hip. The lesions spread slowly over his arms, trunk and legs. There was no itching. He had had some edema of his ankles for three or four weeks and occasional nose bleeds.

Physical Examination.—The eruption consisted of a generalized mottled or reticulated erythema affecting almost the entire body with the exception of the central portion of the chest and back. The color varied from light to dark or purplish red, and in some areas small confluent patches were found which were slightly elevated, tender and often hemorrhagic.

In other locations, particularly on the arms and thighs, the skin in indefinitely outlined areas had assumed a dull, chalky appearance with a slightly thickened feel. We did not determine whether these areas were a part of the central white portion of the reticulated pattern or had developed independently.

There were some petechial hemorrhages in the conjunctivas, blood crusts in the nose and a diffuse reddening of the throat. His spleen and liver were palpable,

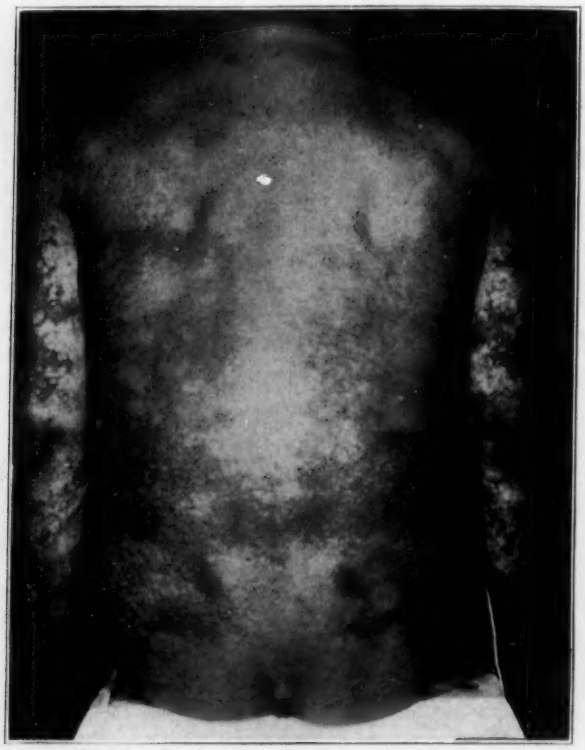

Fig. 1.—Patient with periarteritis nodosa showing extensive livedo reticularis.

Fig. 2.—Right leg of patient. Note swelling of knee.

and there were a few rales at the base of the lungs. His right knee was swollen with some fluid in the joint, and there was edema of the ankles. His temperature was 101.6 F., the pulse rate 96, systolic blood pressure 200 and diastolic 120. During his stay in the hospital the temperature was of the septic type with a daily peak around 102 or 103 F. The blood pressure became stabilized at 220 systolic and 140 diastolic. The blood on first examination showed 3,720,000 red blood cells, with 74 per cent hemoglobin, and 12,500 white blood cells, with 70 per cent polymorphonuclear leukocytes, 3 per cent juvenile neutrophils, 1 per cent eosinophils, 1 per cent basophils, 16 per cent lymphocytes and 9 per cent monocytes. On subsequent weekly examinations it showed an average of 18,000 leukocytes with about 88 per cent polymorphonuclear neutrophils. Four blood cultures made at weekly intervals were negative. The Wassermann reaction of the blood was negative. On first chemical examination the blood showed 5.99 Gm. of total protein per hundred cubic centimeters, 38 mg. of nonprotein nitrogen, 100.2 mg. of serum chlorides and an albumin-globulin ratio of 47 per cent albumin and 53 per cent globulin. The carbon dioxide–combining power of the blood was

Fig. 3.—Legs of patient after ulceration had begun.

57.9 volumes per cent. The nonprotein nitrogen continued to rise and at death was 116 mg. per hundred cubic centimeters. The total proteins remained stationary at 6.16 Gm., but the albumin-globulin ratio continued to reverse itself with a final value of 41 per cent albumin and 59 per cent globulin. The blood calcium was 7.9 mg. per hundred cubic centimeters and phosphorus 7.3 mg. The van den Bergh reaction of the blood was negative. The electrocardiogram was normal.

A heavy trace of albumin, many hyaline and granular casts, pus cells and red blood cells were found in the urine. The phenolsulfonphthalein test for renal function showed 49 per cent at the end of two hours. The urea clearance test for renal efficiency revealed an average of 65 per cent.

Filtrates from seven strains of streptococci injected intracutaneously gave negative reactions.

Roentgenograms of the chest showed fibrosis of both roots extending into the perihilar region, particularly toward the bases. A large calcified Ghon tubercle was present in the right base. Later examination revealed signs of chronic passive congestion and lobular pneumonia.

Course.—On admission the patient had no particular complaint except the rash. Later, however, there were manifestations of delirium, peripheral neuritis,

severe abdominal pain with bloody diarrhea, cardiac decompensation with chronic passive congestion and cough with blood-streaked sputum. The pain in the skin increased until he could hardly bear to have it touched. Deep purplish areas appeared, became necrotic and ulcerated, forming lesions several centimeters in diameter. Toward the end the abdomen became distended with fluid. There was evidence of chronic passive congestion and lobular pneumonia, and the patient became irrational and died seven weeks after admission to the hospital.

The diagnosis was not made before death. We felt at first that it might be an unusual manifestation of acute lupus erythematosus.

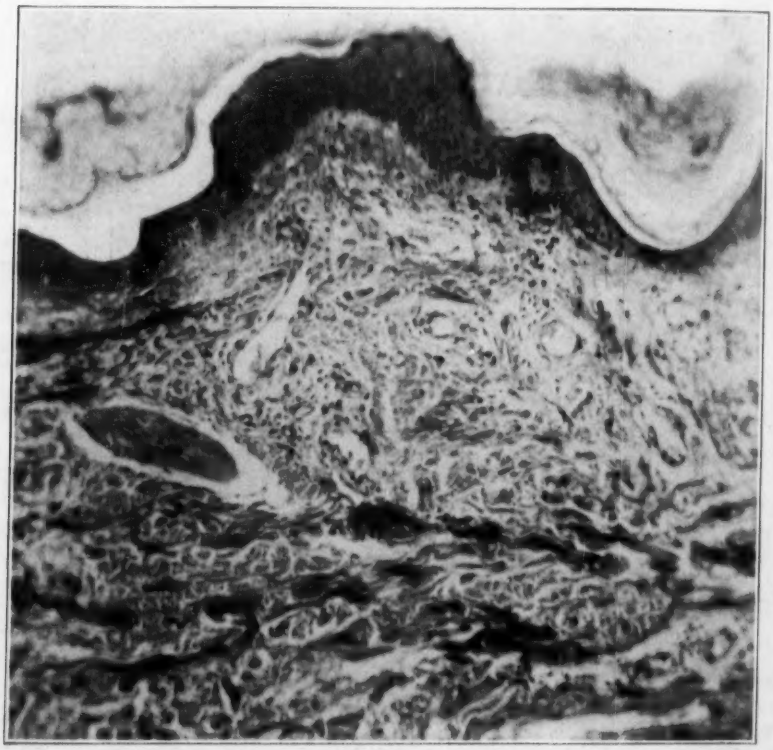

Fig. 4.—Biopsy specimen 1, showing dilatation of the small blood vessels and a mild perivascular infiltration in the upper portion of the cutis.

Dr. Longcope thought, however, that with the elevation in blood pressure and the changes in the urine, one must conclude that the patient had hemorrhagic nephritis, probably in the acute stage, and that the cutaneous changes might correspond with the so-called erythema group associated with acute nephritis.

Anatomic Diagnosis.—After the autopsy the diagnosis was: periarteritis nodosa of the heart, kidneys, stomach, mesentery, rectum, skin and pulmonary artery, with myocardial scarring, cirrhosis of the liver, diphtheritic colitis, pulmonary edema and lobular pneumonia, thrombosis of a branch of the middle cerebral artery, hemorrhages in the mucosa of the bladder and enlargement of the mesenteric lymph nodes.

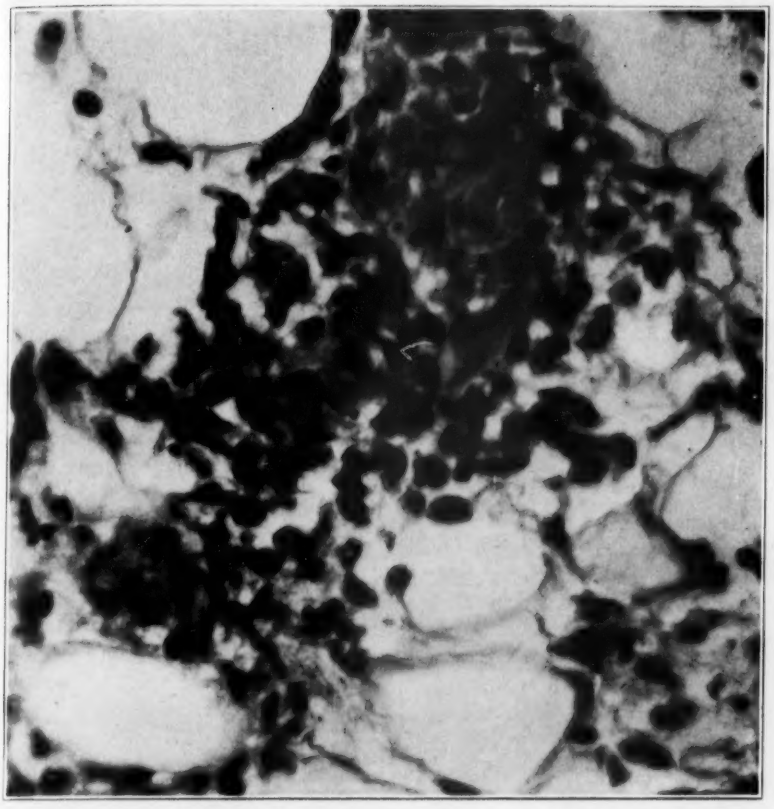

Fig. 5.—Biopsy specimen 1, showing dense perivascular infiltration and thrombosis of the small vessels in the fatty layer.

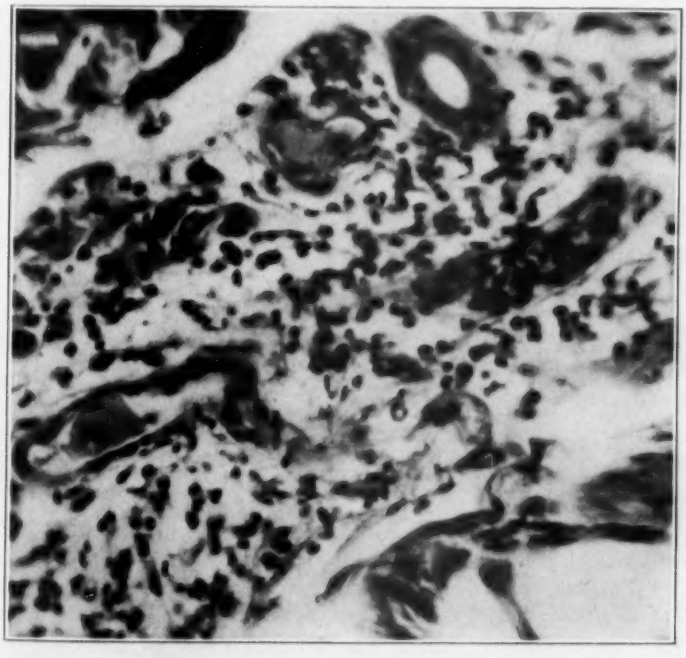

Fig. 6.—Biopsy specimen 1. Thrombi may be seen in two of the small vessels, and another one is choked with leukocytes.

Postmortem Examination (Dr. MacCallum).—The organs showed in many places the most typical and striking lesions of the nature of periarteritis nodosa. Such changes in the small arteries were seen especially in the heart, stomach, intestine and kidney, although a few were found elsewhere. None were seen about the adrenal glands.

Various stages were recognizable, and where they were best distinguished it seemed that necrosis with hyaline alteration, which stained like fibrin, was found as a layer between the outer layer of the intima and the inner layer of the media. Later this was replaced by a new tissue of elongated cells, with many small round cells, and such a tissue was also found in a thick layer outside the adventitial remains. Such changes leading to obstruction of some of the vessels produced areas of necrosis in the kidney. There were also more diffuse changes there suggesting a separate glomerulonephritis. The glomeruli showed thickened capillary tufts and crescents in many cases.

The heart was scarred, evidently from older changes. The pulmonary artery was thickened, its fibers were spread apart, and there were nodules of the characteristic type on small vessels in the adventitia. The liver showed a peculiar change: there was widening and scarring of the tissue about the portal spaces, but this condition gave the impression of being due not to a loss of liver cells but rather to a great infiltration of mononuclear cells. The spleen showed a marked accumulation of cells between the sinuses, as in acute splenic tumor, although it did not appear soft or bulging at autopsy.

Histologic Examination.—Specimen 1 was removed from a bluish red net of the reticulated pattern on the thigh. The epidermis presented no noteworthy changes (fig. 4). In the upper third of the cutis the small blood vessels were widely dilated and surrounded by an edematous tissue which showed some proliferation of the fixed cells and occasionally a few polymorphonuclear leukocytes. There were no definite changes in the vessel walls or formation of thrombi. In the lower portion of the cutis, however, particularly between the coils of the sweat glands and in the fat columns, were areas where the small blood vessels were surrounded by a variable infiltration of leukocytes, with frequently many eosinophils, which often invaded the walls and filled the lumens (figs. 5 and 6). In other instances the vessels were occluded by thrombi, which also were found where there was little or no perivascular infiltration (fig. 7). The walls of such vessels were usually intact but occasionally showed degenerative changes (fig. 8). The thrombi were formed from broken-up red blood cells, either with or without nuclear detritus, and many of them were converted into a hyalin-like homogeneous bluish-staining material. Whether or not there was intimal proliferation was difficult to determine because the affected vessels were, on the whole, too small to show conclusively the progressive vascular changes characteristic of this disease. The specimen contained three or four fair-sized arteries, but none of these were involved. The elastic tissue appeared normal, and the sweat glands presented no definite degenerative changes.

Specimen 2 was removed from one of the chalky white areas on the thigh. The epidermis was atrophic (fig. 9). In the upper portion of the cutis, in contrast to specimen 1, there were few dilated blood vessels, but they seemed to have been replaced for the most part by fibrous cords, frequently with atrophic nuclei, in which definite lumens were difficult to demonstrate. The nuclei of the collagenous tissue were also frequently atrophic. In the lower half of the cutis there were some dilated vessels, and a few showed degenerative changes, intimal proliferation or formation of thrombi. Here, however, there was little leukocytic infiltration, but the vessels were surrounded by or lost in richly cellular connective tissue in

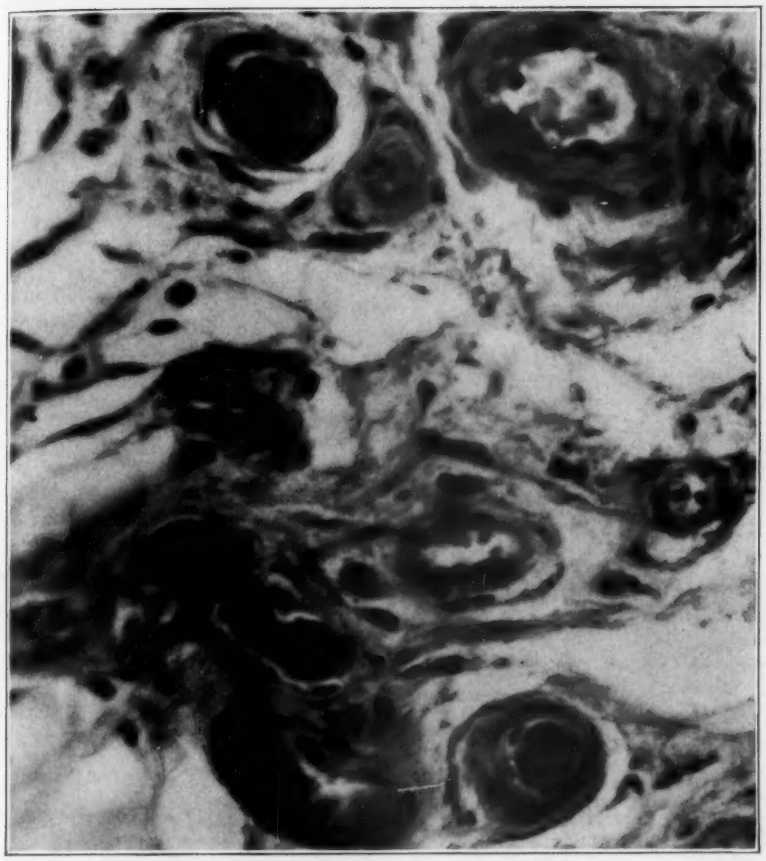

Fig. 7.—Biopsy specimen 1. Most of the small vessels are filled with thrombi. There is little perivascular infiltration in this area.

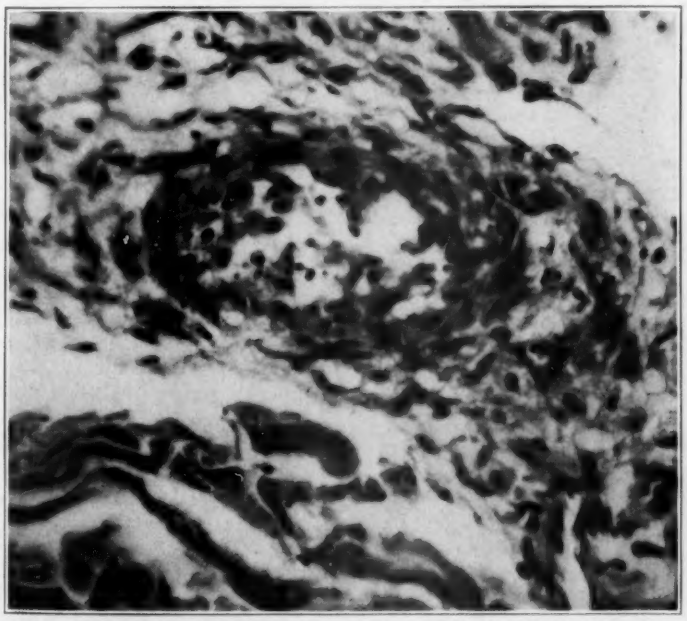

Fig. 8.—Biopsy specimen 1, showing degeneration of the media of a small artery and a lumen partially filled with cellular debris.

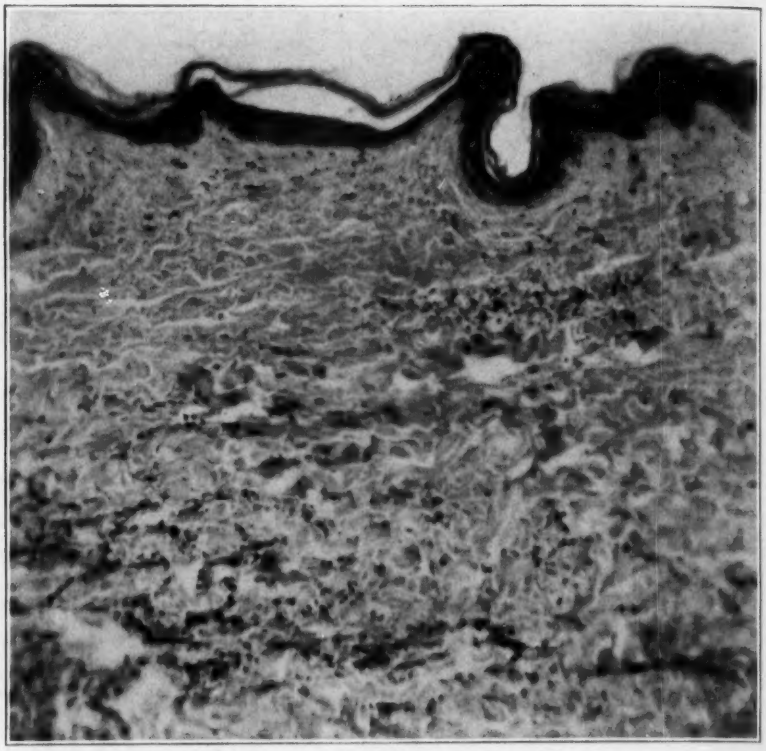

Fig. 9.—Biopsy specimen 2, showing decided atrophy of the epidermis and cellular tissue in the upper part of the cutis. The process in this portion of the section is somewhat more exaggerated than it is in the specimen as a whole.

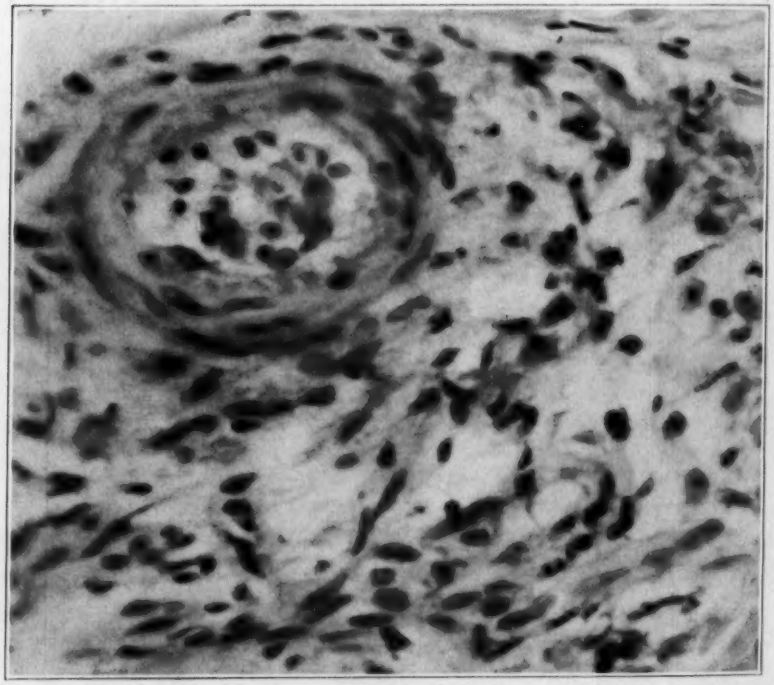

Fig. 10.—Biopsy specimen 2. In the deeper layers of the cutis is seen proliferation of the young connective tissue cells with only a sprinkling of leukocytes. The small artery in the picture shows some intimal proliferation, which may be interpreted as representing Arkin's granulation tissue stage, in contrast with figures 4 to 8 which would correspond to the acute inflammatory stage.

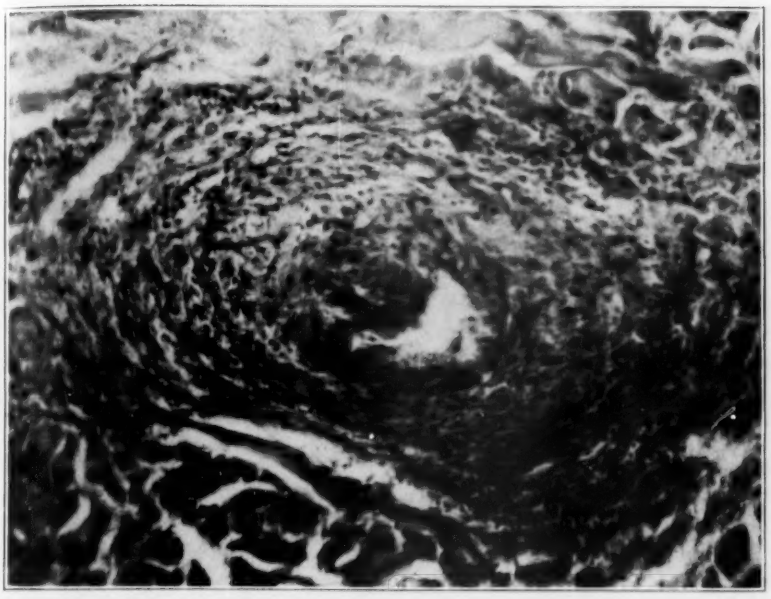

Fig. 11.—Biopsy specimen 3. This artery showed intense infiltration of the media and periadventitial tissues with leukocytes and red blood cells. The irregular mass in the lumen is made up mainly of well preserved red blood cells.

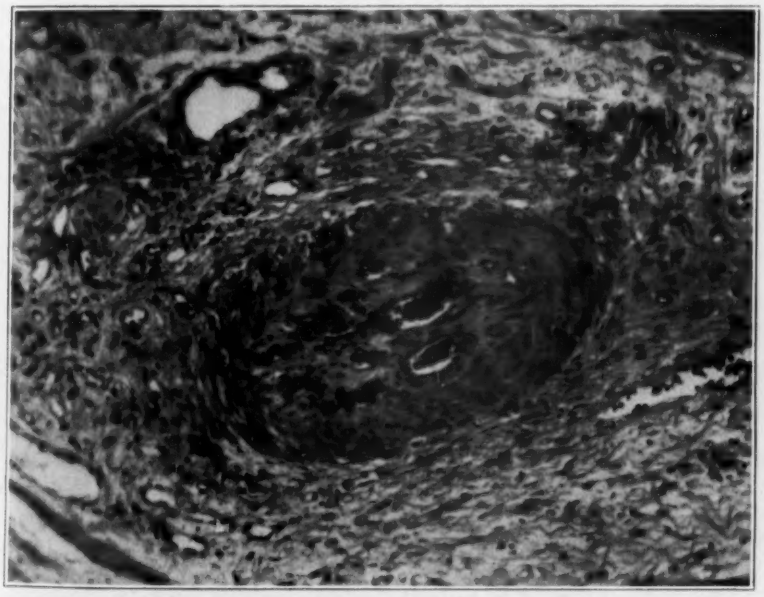

Fig. 12.—Biopsy specimen 4, showing a canalized artery representing the healed stage of the disease.

which some hemorrhages were seen (fig. 10). In some cases the sweat glands were atrophic or had been displaced by young connective tissue. The elastica seemed to be intact.

Specimen 3 was taken at autopsy and included an area that showed ulcer formation. It was much larger and deeper than the biopsy specimens. Over the nonulcerated portion the epidermis was either normal in appearance or acanthotic. In the upper portion of the cutis the blood vessels in general were widely dilated and occasionally contained degenerated red blood cells and leukocytes. Around the base of the ulcer most of them were filled with thrombi. In the deeper portion of the cutis there was considerable perivascular infiltration, and some of the vessels showed degenerative changes in their walls with or without thrombus formation. In the fatty tissue there was, in some locations, well marked *Wucheratrophy;* in others the capillaries were choked with leukocytes. Here also thrombi were occasionally seen. There were numerous comparatively large arteries in the subcutaneous tissues, but only one of these showed the characteristic changes of periarteritis nodosa, although some of the smaller vessels were affected (fig. 11). There were hemorrhages here and there, and in one area a large collection of phagocytes filled with pigment was seen.

Specimen 4 was taken from a partially ulcerated area on the scrotum. Here the most striking thing was the intense edema with numerous widely dilated vessels. The majority of the vessels were unaffected except for a variable perivascular infiltration. A few of them, however, showed considerable infiltration of their walls with occasional necrosis, but no definite thrombi were encountered except in the base of the ulcer. Only one of the fairly large arteries showed the characteristic changes of periarteritis nodosa (fig. 12). This lay in the lower third of the cutis, was surrounded by fibrous tissue and had become canalized.

COMMENT

Periarteritis nodosa, if judged by the number of reported cases, is rare, but this is perhaps more apparent than real, because in the majority of instances the diagnosis is made at autopsy. Bernstein [12] in 1933 published a report of a case in which the nature of the disease was revealed before death by a muscle biopsy. He stated that he was unable to find the report of any case without peripheral nodules in American or English literature in which the diagnosis had been so made. Because of the lack of any coordinated general symptoms or physical signs characteristic of the disease the cutaneous manifestations offer an obvious cause for suspicion and an easily accessible means of diagnosis, particularly when nodules are present. It is doubtful, however, that the diagnosis could be made always from the more superficial erythematous or papular eruptions, because most of the blood vessels in the upper layers of the cutis are too small to show conclusively the progressive vascular changes characteristic of the disease.

Our first two biopsy specimens, although they included a small amount of the subcutaneous fat, contained only two or three fairly large

12. Bernstein, A.: Periarteritis Nodosa Without Peripheral Nodules Diagnosed Antemortem, Am. J. M. Sc. **190**:317 (Sept.) 1935.

arteries and these were unaffected. Thrombus formation, however, with or without infiltration of the vessel wall, and occasionally necrosis were common in the smaller vessels and should make one at least suspicious that this condition was present. Our autopsy specimens, which were unusually large and included the entire thickness of the subcutaneous fat and some of the muscle, contained numerous relatively large arteries, but we found only two of these that showed the characteristic changes. Many of the smaller vessels and capillaries, however, in the lower portion of the cutis as well as in the subcutaneous fat were thrombosed and showed inflammatory changes in or around the walls. We were not able to determine in all cases whether these changes affected veins or small arteries but were under the impression that both were involved, the veins, however, less frequently.

The exact mechanism of mottling of the skin is not known, but Lewis [13] stated the belief that the evidence at hand points to a correspondence between the areas of mottling and the main arteriolar territories and that it is due to a variation in the blood supply in which the pale central portions of the pattern represent the areas of maximum flow. In the marginal zones, where the flow is somewhat less because of a lack of tone in these vessels, any impediment to the blood supply, either a spasm of the small arteries or occlusive inflammatory processes, might well result in an accentuation or a permanence of this pattern. The condition is of congenital origin and in its mildest form, the so-called cutis marmorata, it is transitory and is brought out most strikingly by cold. In other cases the pattern is permanent, either because the congenital vascular anomaly is developed to a great degree or because some disease affecting the blood vessels has accentuated this tendency.[14] In the latter group tuberculosis and syphilis have been particularly mentioned, and often pathologic alterations in the blood vessels have been demonstrated. The more or less permanent forms are referred to as livedo reticularis or livedo racemosa when the lesions have more of a treelike shape and their branches are raised or palpable.

We were not able in our case to determine the exact relation between the whitish chalky areas on the skin and the reticulated pattern. The natural assumption would be that these areas corresponded to the normal

13. Lewis, T.: The Blood Vessels of the Human Skin, and Their Responses, London, Shaw & Sons, Ltd., 1927.

14. Williams, C. M., and Goodman, H.: Livedo Reticularis, J. A. M. A. **85**:955 (Sept. 26) 1925. Becker, S.: Generalized Telangiectasia: Clinical Study, with Special Consideration of Etiology and Pathology, Arch. Dermat. & Syph. **14**:387 (Oct.) 1926. Ebert, M. H.: Livedo Reticularis, ibid. **16**:426 (Oct.) 1927. Barker, N. W., and Baker, T. W.: Proliferative Intimitis of Small Arteries and Veins Associated with Peripheral Neuritis, Livedo Reticularis and Recurring Necrotic Ulcers of the Skin, Ann. Int. Med. **9**:1134 (Feb.) 1936.

central pale zones, but if such zones represent points of maximal blood supply this conception on first thought would hardly be acceptable, particularly since the specimen removed for biopsy in one of these areas certainly showed evidence of a decided diminution of the blood supply.

It is possible, however, that they may have represented the healing or granulation tissue stage of the disease and this explanation is supported to some extent by the histologic observations. Such areas may have passed through the inflammatory stage without suffering from a sufficient vascular occlusion to cause ulceration, and in this case they would be most likely to survive if they had corresponded to points of maximal blood supply in the skin. It was the opinion of the medical staff who followed this patient during his illness that ulceration began in the reddish cyanotic tender zones and not in the ischemic locations.

SUMMARY

A case of periarteritis nodosa is reported in which cutaneous manifestations were present, consisting of livedo reticularis followed by necrosis and ulceration.

In the biopsy specimens taken during life the small blood vessels showed changes which, if not absolutely diagnostic, made one suspicious that the disease was periarteritis nodosa. In the much larger and deeper autopsy specimens of the skin, however, a few of the larger arteries were characteristically affected.

In a review of the literature it was found that some sort of cutaneous manifestation had been noted in about 25 per cent of the cases. In 3 cases besides our own there was livedo racemosa or livedo reticularis.

We have emphasized the importance of a more general recognition of the cutaneous manifestations of periarteritis nodosa, principally because, from the standpoint of diagnosis, the skin is the most accessible portion of the body for histologic study.

ABSTRACT OF DISCUSSION

DR. WILLIAM H. GOECKERMAN, Los Angeles: I should like to ask Dr. Ketron what particular features of the clinical and histologic examination he would emphasize as distinctive and of particular value in contributing to the diagnosis.

DR. LLOYD W. KETRON, Baltimore: It is generally known that there is no train of symptoms on which the diagnosis of periarteritis nodosa can be made absolutely. The final diagnosis must rest on the pathologic changes, which are usually characteristic. In such cases as ours, however, in which no cutaneous nodules are found, I doubt whether one could make the diagnosis from the usual sections of skin, although the disease should certainly be suspected. In our case thrombus formation was the most outstanding change in the cutis. I have never seen it so strikingly developed except in erythema pernio. Of course, in large and deep biopsy specimens which include the subcutaneous tissue the characteristic changes in the larger arterioles would probably be found in many instances.

NAEVO-EPITHELIOMA ADENOIDES (CYLINDROMA) OF THE SCALP

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Multiple cylindroma or true turban tumors of the scalp are relatively rare. Ronchese [1] reviewing the literature on turban tumors to 1932 could collect only 31 fully developed cases. Since 1932 Schuermann and Weber [2] have reported 5 cases which they observed in the clinic of Friboes, thus bringing the total number of reported cases to 36. Most of the cases reported are from German and French literature. Only 5 observations are fully recorded in the American literature. The American cases were reported by Ronchese,[1] Cohn,[3] Sutton,[4] Stillians [5] and Jones, Alden and Bishop.[6] Hence, the report of an additional case with classic clinical and histologic findings is justified.

REPORT OF A CASE

History.—D. L., a Jewish man aged 68, born in Lithuania and resident in America for many years, came to the skin dispensary of the Mount Sinai Hospital on March 12, 1939, for treatment of multiple tumors of the scalp. Two tumors were excised for histologic study, and he was referred to surgeons for further excision of the bleeding and fungating tumors.

The patient had been in good health throughout his adult life. About twenty-five years ago, while he was working as a plumber, his scalp was torn, and he stated that he believed that the growths came as a result of that injury. His only complaints were of the unsightly appearance of his head and the frequent bleed-

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From the Department of Dermatology, Mount Sinai Hospital, service of Dr. Maurice Dorne.

This case was presented at a meeting of the Chicago Dermatological Society on March 15, 1939.

1. Ronchese, F.: Multiple Benign Epithelioma of the Scalp (Turban-Tumors), Am. J. Cancer **18**:875 (Aug.) 1933.

2. Schuermann, H., and Weber, K.: Beitrag zur Kenntnis der Spieglerschen Tumoren (Cylindrome) nebst einigen Bemerkungen zum Epithelioma adenoides cysticum, Arch. f. Dermat. u. Syph. **175**:682, 1937.

3. Cohn, I. E.: A Case of Multiple Sarcoma, J. Cutan. & Genito-Urin. Dis. **10**:393, 1892.

4. Sutton, R. L.: Diseases of the Skin, ed. 8, St. Louis, C. V. Mosby Company, 1931.

5. Stillians, A. W.: Nevo-Epithelioma Adenoides (Cylindroma) of the Scalp, Arch. Dermat. & Syph. **27**:481 (March) 1933.

6. Jones, J. W.; Alden, H. S., and Bishop, E. L.: Turban Tumor, or Sweat Gland Carcinoma; So-Called Epithelioma of the Scalp; Report of a Case Illustrating Its Epithelial Structure, Arch. Dermat. & Syph **26**:656 (Oct.) 1932.

ings. His daughter stated that the fungating tumors were emitting a foul odor. There was no history of a similar condition in any other member of his family. In 1922 about fifty tumors and nodules of the scalp were excised, but most of them have since recurred.

Physical Examination.—On the face and forehead were many discrete pinhead-sized whitish papules with the clinical appearance of milia. At the hair line of

Fig. 1.—Appearance of tumors of scalp in 1922.

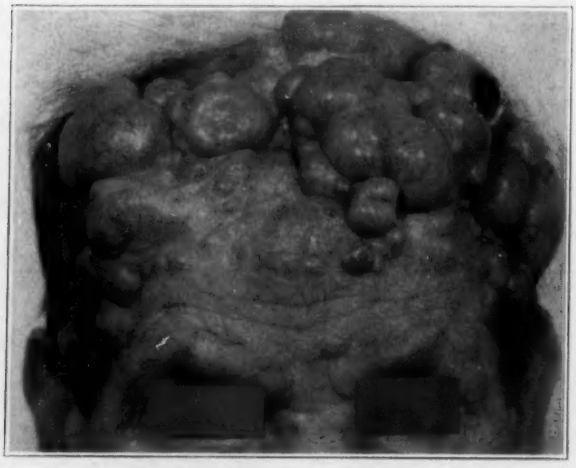

Fig. 2.—Appearance of tumors of scalp in 1939.

the scalp and on the frontal, parietal and occipital areas of the scalp were clusters of tumors, about ninety, ranging from the size of a navy bean to that of a tomato. Some of these tumors were protruding, lobulated and freely movable. Some were pedunculated with a narrow base, while the majority were fixed to the scalp. On the chest and on the back were a number of scattered soft protruding bluish tumors. These were painless and covered with normal skin. They varied in size from that of a small pea to that of a hazelnut. The tumors of the scalp were pink. They were covered with a thin cigaret paper-like epidermis. They showed enlarged

tortuous vessels. They felt rubbery to the touch and were smooth and hairless. A few of the tumors showed central or eccentric erosions which were covered with a sanguinous crust. One tumor was fungating and discharged a foul odor.

Pathologic Observations.—The excised tumors were examined by Dr. I. Davidschn, pathologist of the Mount Sinai Hospital, who gave the accompanying report.

"The specimen is a nodular mass measuring 20 by 18 by 7 mm. One surface is covered with skin, the other is bare and shows connective and fat tissue. The skin appears atrophic. It is covered with many grayish black hairs, which are growing particularly from the periphery. The consistency is moderately firm. The cut surface is homogeneously yellowish gray and nodular (fig. 3). The surface is covered with a thin stratified and hornifying squamous epithelium. The papillary layer is atrophic. The tissue underneath is loose and cell poor, with many hair follicles, occasional sebaceous glands and a moderate, mainly perifollicular infiltration with lymphocytes. Deeper down large masses of cells

Fig. 3.—Biopsy specimen.

are present, varying in size, with some confluent, which fill out the alveolar spaces. These accumulations vary considerably in shape; some are round, some oval, some tubular and some entirely irregular. They resemble a nevus in shape and in arrangement. The nodules are enclosed in a homogeneous outer lining of pink. This outer casing varies in thickness from a hardly visible layer to a wall several microns thick. A fine network extends from this outer casing into the lumen and separates the cells from each other. In some places the appearance and tinctorial characteristics are the same as those of the outer casing. Some of the sweat glands at the periphery of the tumor are surrounded by a casing similar to that which encloses the tumor alveoli (fig. 4). There are two distinct varieties of cells. One variety, found at the periphery adjacent to the casing, have dark, hyperchromatic oval or elongated nuclei. In some places one sees the same type of cells along the intra-alveolar extensions of the outer casing. The other cells have pale vesicular nuclei and occupy the bulk of the alveolar spaces. They have a small amount of cytoplasm, partly vacuolated. Mitotic figures are very rare. Occasional cystic, geometrically round spaces are seen, enclosed by tumor cells.

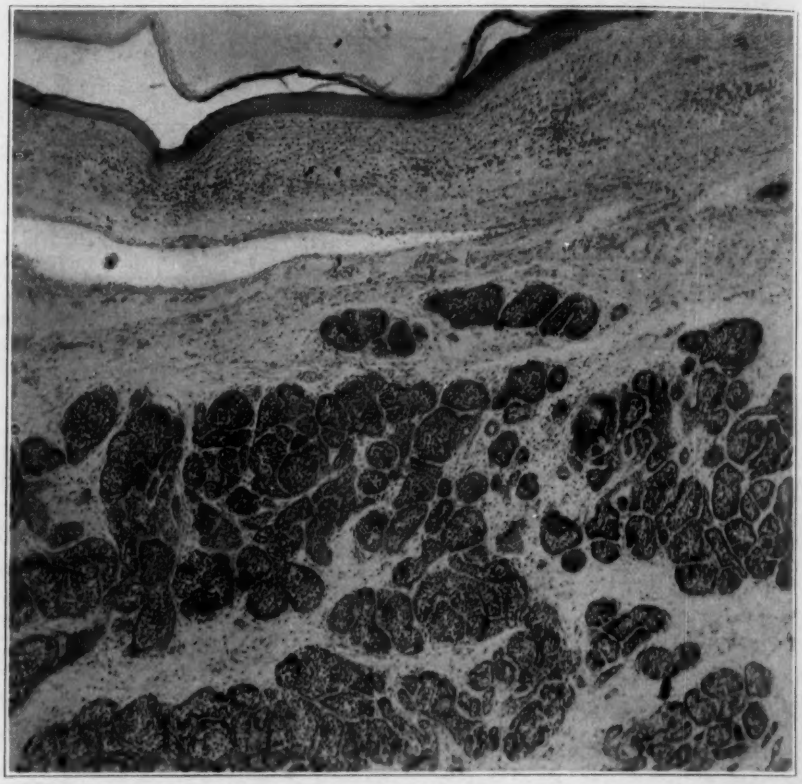

Fig. 4.—Low power magnification showing the masses of the tumor cells. Note the location, size, shape and arrangement (× 53).

Fig. 5.—Detail of cylindroma showing the round cystic spaces and the epithelial masses surrounded by the thick outer casing (× 80).

Some are filled partly with hyalinized spheric bodies, and some are filled with varying amounts of shrunken pink-staining granular material, but most are empty. They are lined with a homogeneous thick pink-staining layer, resembling the outer casing. Outside of it one sees in some of them cuboidal epithelium-like cells and in others flat endothelial-like cells. The stroma is scanty, loose and cell poor (fig. 5). The picture is that of a basal cell epithelioma of the type known as cylindroma. The term "naevo-epithelioma adenoides" is most appropriate for these tumors from the standpoint of what is known about their histogenesis. They are known to be benign."

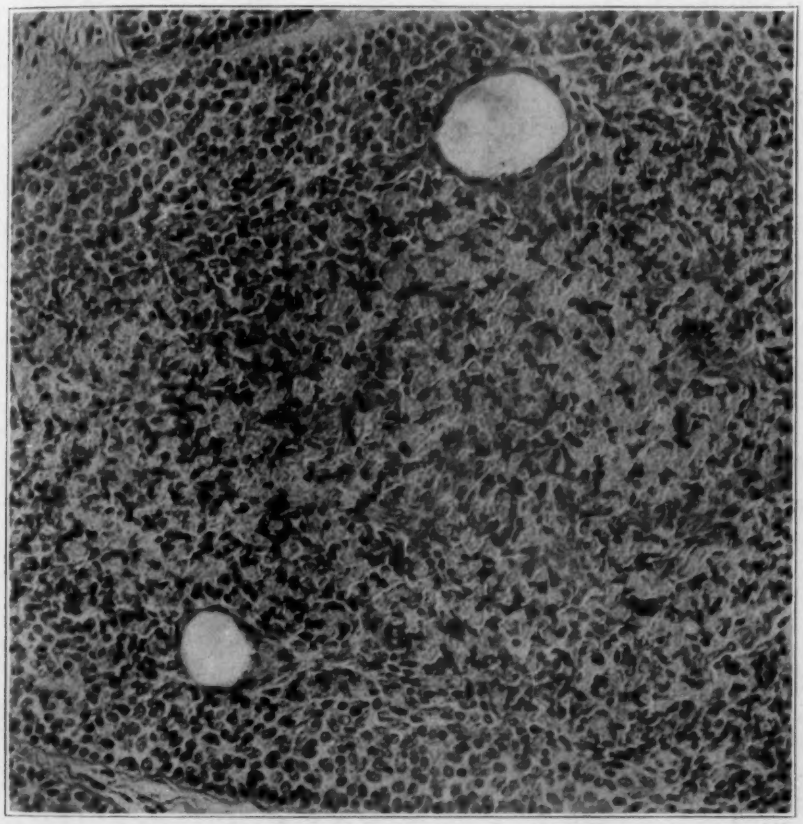

Fig. 6.—High power view to illustrate the detail of the cysts (× 270).

SUMMARY

A classic case of multiple cylindroma or turban tumors of the scalp is reported, in which: (1) there was no familial history of such a condition; (2) a history of trauma preceded the appearance of the growths, and (3) some of the growths were fungating but were histologically benign.

The term suggested by E. Hoffman for these tumors and used by Stillians,[5] "naevo-epithelioma adenoides," seems to be most appropriate.

SMALL NODULAR NONCASEATING TUBERCULODERM

MODES OF INFECTION AND LOCALIZATION, WITH REPORT OF A CASE

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The clinical and histopathologic features of cutaneous tuberculosis are dependent on the balance struck between the resistance and sensitivity of the host and the virulence and number of the invading organism (Mycobacterium tuberculosis). A certain degree of inherent resistance exists in the tissues of the normal person, which is gradually augmented over a period of years after primary infection, a hypothetic immunizing response to tubercle bacilli known as acquired immunity. While primary infection is relatively innocuous in children, an exogenous reinfection is liable to be an activating process (superinfection) if the acquired immunity is not sufficiently developed. Tuberculous infection is a constant liability, whether it is active, inactive or "healed" by fibrosis and calcification or by resorption, as extension and dissemination may occur at any time the resistance is weakened and endogenous reinfection results. The longer the mechanism of acquired resistance is operative, the greater the immunity of the subject to tuberculous infection, which explains why adults who have had small healed foci or small reinfections for many years have greater resistance than have children or young adults whose tuberculization is recent.

Tuberculoallergy, an acquired sensitization to the protein of tubercle bacilli, has its initiating phase in the primary complex, when phagocytosing monocytes liberate the bacillary protein. Sensitization is induced by the tubercle bacillus itself and cannot be brought about in the normal person by injection of tuberculin alone, so that there is something more than the bacillary protein thus far known to reside in the bodies of tubercle bacilli (living or dead) which sensitizes the person. Sabin and Joyner [1] have shown that synergic sensitization of animals by intra-

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From the Children's Hospital.

Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 3, 1939.

1. Sabin, F. R., and Joyner, A. L.: Tubercular Allergy Without Infection, J. Exper. Med. **68**:659 (Nov.) 1938.

dermal injection of combined tuberculoprotein and tuberculophosphatide produces sensitivity identical with that induced by tubercle bacilli. The tuberculin reaction may or may not be related to the biologic immune process, and, while it usually shows the degree of tuberculosensitivity, it is not indicative of the immune status. Rich [2a] stated the belief that tuberculoallergy is a process distinct from resistance or the immune reaction. Krause, Zinsser and others concluded that it is a phase of immunity. Tuberculin therapy reduces tuberculohypersensitivity, allowing greater freedom to the immune antituberculosis reaction for resolution and healing. Sensitivity to tuberculin may decrease as immunity increases, and if tuberculoallergy is abolished by desensitization to tuberculin the immunity persists.[2b]

The virulence of tubercle bacilli consists primarily in their ability to multiply in the tissues and is in direct relation to the potency or weakness of the immune reaction, which determines the tendency toward resolution or extension of the tuberculous process. The number of living bacilli locally influences the character of the lesion.

MODES OF INFECTION AND LOCALIZATION

Tuberculoderms are the result of (*a*) local primary or secondary inoculation, (*b*) lymphogenous localization or (*c*) hematogenous dissemination of tubercle bacilli.

Tuberculoderms Due to Primary and to Secondary Inoculation.—Primary inoculation occurs when the patient has never been tuberculized; secondary inoculation is a reinfection and occurs when the patient has been previously tuberculized. Tubercle bacilli may enter the skin by exogenous inoculation, causing the primary tuberculous "chancre" and complex (enlarged regional glands) in children who have never been tuberculized or tuberculosis luposa (wound infection or secondary inoculation lupus) in children who have been tuberculized. Exogenous inoculation in a tuberculized adult results in tuberculosis verrucosa or tuberculosis fungosa and becomes a superinfection if active visceral tuberculosis is further activated. Endogenous secondary autoinoculation by the patient's own saliva containing viable tubercle bacilli may result in tuberculosis verrucosa, while the excretions from active foci of tuberculosis of the respiratory, the intestinal or the genitourinary tract may result in tuberculosis ulcerosa orificialis.

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2. (*a*) Rich, A. R., and McCordock, H. A.: An Inquiry Concerning the Role of Allergy, Immunity and Other Factors of Importance in the Pathogenesis of Human Tuberculosis, Bull. Johns Hopkins Hosp. **44**:273, 1929. (*b*) Rich, A. R.; Jennings, F. B., and Downing, L. M.: The Persistence of Immunity After the Abolition of Allergy by Desensitization, ibid. **53**:172, 1933.

Lymphogenous Tuberculoderms.—Tuberculosis luposa in the majority of cases is probably due to secondary exogenous implantation or to reinfection of the nasal mucosa in childhood by direct infection from the fingers with lymphatic extension to the face and to the nasal or buccal mucosa, where the lupous lesion develops in the lymph spaces of the cutis and stroma respectively, its further local advance occurring by contiguous lymphatic extension. Tuberculosis luposa may develop from lymphatic extension of tuberculosis colliquativa or tuberculosis verrucosa. In the majority of cases tuberculosis colliquativa is probably due to secondary exogenous implantation or reinfection of the pharyngeal lymphoid tissue in childhood with lymphatic extension to the neck where the scrofuloderm develops in the cutaneous or subcutaneous lymphatic vessels. The lesions occurring in other regions, especially in adults, are due to lymphatic extension from a tuberculous gland, bone or joint. Tuberculosis fungosa occurs in adults and is due to lymphatic extension from a focus of tuberculosis verrucosa or from tuberculous bone, if not from secondary exogenous inoculation.

Hematogenous Tuberculoderms.—Though tubercle bacilli have been found in the blood, their demonstration is exceedingly difficult, as they are rapidly disposed of when present. Tubercle bacilli or monocytes containing tubercle bacilli may enter small arteries and veins repeatedly from caseating pulmonary tuberculous foci, with hematogenous metastases to the cutaneous capillaries. Several of the clinical varieties of cutaneous tuberculosis are believed to be blood borne from subclinical visceral, glandular, osseous or seromembranous tuberculous foci, but these are usually difficult to discover. Occasionally small fibrosed or calcified foci of "healed" tuberculosis in the lungs or glands are suspected, or active tuberculous fibrosis in the lungs is discovered. Calcification of the lesions of tuberculous adenitis cannot be considered to represent healing, as reactivation may occur years later, according to Duken.[3] Myers and Harrington [4] stated the belief that tubercle bacilli may remain alive and virulent for many years in encapsulated and calcified pulmonary foci, later resorption of the calcification freeing the formerly imprisoned bacilli and causing contiguous extension of the tuberculous process. Hematogenously disseminated tubercle bacilli lodging in the cutaneous capillaries are probably phagocytosed by the endothelial lining cells and carried through the vessel wall into the perivascular lymph spaces or (by monocytes) into the lymph spaces of the cutis, where a typical tubercle or a tuberculoid reaction occurs by

3. Duken, J.: Ueber Verlaufsarten der extrapulmonalen Primärtuberkulose, Ztschr. f. Kinderh. **55**:687, 1933.

4. Myers, J. A., and Harrington, F. E.: The Effect of Initial Tuberculous Infection on Subsequent Tuberculous Lesions, J. A. M. A. **103**:1530 (Nov. 17) 1934.

accumulation of wandering monocytes at the site. Actual bacillary emboli may suddenly occlude the vessel, resulting in anemic tissue necrosis and ulceration with a nonspecific inflammatory reaction before the bioimmune monocytic reaction has had an opportunity to occur. Ischemic tissue necrosis and ulceration may occur in lesions located on acrocyanotic regions.

Tuberculoderms due to hematogenous dissemination of tubercle bacilli are tuberculosis luposa disseminata, tuberculosis miliaris disseminata, acne scrofulosorum and tuberculosis lichenoides, which occur in children with underdeveloped immunity, and tuberculosis miliaris faciei, rosacea-like tuberculid, tuberculosis papulonecrotica (acnitis and folliclis) and tuberculosis indurativa (erythema induratum), which occur in adults with weakened immunity.

NECROSIS AND CASEATION IN TUBERCULODERMS

In the tuberculous granuloma the monocytes phagocytose the tubercle bacilli, and if the cellular antibody reaction is sufficient the bacilli are destroyed by granular degeneration and the monocyte is metamorphosed to an epithelioid cell. Mature epithelioid cells may signify dead tubercle bacilli. In the process of digesting bacilli the monocyte or epithelioid cell may undergo necrosis or granular degeneration if the antibody formation is insufficient to overcome the cytotoxic action of the bacilli. Centrally placed epithelioid cells in the tubercle may undergo mass necrosis and fuse into microscopic areas of caseation from loss of their cellular surface tension. Caseation occurs more rapidly when there are cellular hypersusceptibility to tuberculoprotein and a lowered resistance mechanism. Giant cells of the Langhans type are formed by the fusion of degenerated phagocytosing monocytes which have lost their surface tension, while giant cells with centrally clustered nuclei may be due to nuclear multiplication. Anemic tissue necrosis either from embolic occlusion of the vessels or occlusion due to compression occurs with nonspecific inflammatory cellular reactions and may be augmented if it occurs on acroasphyxial regions of the skin. The purely epithelioid tubercle, which signifies high resistance and tubercle bacilli of low virulence and small numbers, commences as a monocytic infiltrate the cells of which phagocytose the local tubercle bacilli and convert to epithelioid cells. The epithelioid tubercle usually runs a noncaseating course, and while it may be regarded as an abortive tubercle, it frequently shows a narrow peripheral zone of lymphocytes. The typical tubercle with a wide zone of lymphocytes peripherally, which, according to Sabin, signifies resistance, may show latent cellular necrosis.

Although tuberculosis luposa shows no caseation, the older lupous tubercle shows considerable faintly staining areas of granular degeneration or cellular necrosis obliterating the cellular structure together with

the collagen, elastin, glands and blood vessels. Tuberculosis colliquativa shows "creamy liquid" caseation, while tuberculosis miliaris acuta disseminata and tuberculosis ulcerosa orificialis may show actual "cheesy" caseation prior to discharge of the caseous substance by ulceration. Tuberculosis miliaris faciei, tuberculosis verrucosa and tuberculosis fungosa show caseation microscopically. Tuberculosis papulonecrotica and tuberculosis indurativa show anemic tissue necrosis from vascular obliteration by occlusion or by extravascular compression.

The purely epithelioid tubercle is occasionally seen in early tuberculosis luposa disseminata and tuberculosis lichenoides and always in miliary lupoid (Boeck's sarcoid) and lupus pernio. While miliary lupoid is a noncaseating epithelioid tubercle generally believed to be due to hematogenously borne avirulent or nonviable tubercle bacilli, cellular necrosis may occur in rare instances. Caseation and noncaseation are not due to separate types of tubercle infection but are simply phases in the tuberculous process. The hypodermic sarcoid of Darier and Roussy usually presents a noncaseating tuberculoid reaction with giant cells and fibrous trabeculization in the subcutis, in which instances of ulceration have been recorded. It may be a variant of tuberculosis indurativa, but that it is caused by tubercle bacilli is still unproved. Lupus pernio is a broad, flat lesion representing an epithelioid reaction in the cutis of acroasphyxiated areas. It often shows cellular and ischemic necrosis with ulceration.

Tuberculoderms with typical tuberculous histologic changes without caseation are Lewandowsky's rosacea-like tuberculid, Wende's nodular tuberculosis of the hypoderm, some cases of tuberculosis lichenoides (lichen scrofulosorum) and an occasionally noncaseating primary type of tuberculosis miliaris disseminata which disappears by resorption.

Wende's [5] case may be regarded as one of a large nodular noncaseating tuberculoderm in the subcutis. A woman aged 43 had "indurated masses from pea to a pigeon egg in size, deeply under the skin, freely movable, with a cartilaginous border and painful to touch and with no color change" over the right cheek. The tuberculin reaction was positive. Microscopic examination showed nests of epithelioid cells surrounded by round cells with numerous lymphocytes and giant cells in the hypoderm. Subcutaneous fat replacement fibrosis was present. Acid-fast bacilli were present, and animal inoculation gave positive results for tuberculosis. The lesions disappeared by resorption and, as there was no evidence of constitutional tuberculosis, the condition was believed to be of local origin.

Tuberculosis miliaris acuta disseminata is a blood-borne tuberculoderm showing typical tuberculous histologic structure in the cutis, with

5. Wende, G. W.: Nodular Tuberculosis of the Hypoderm, J. Cutan. Dis. **29**: 1 (Jan.) 1911.

caseation. It occurs in infants and children with undeveloped resistance who show a hypoergic or an anergic reaction to tuberculin and is usually fatal. The eruption may be primary, i. e., part of acute visceral miliary tuberculosis, tuberculous meningitis or tuberculous adenopathy, or it may be secondary to an exanthem, such as measles or scarlet fever, in a subject with visceral miliary tuberculosis. The eruption appears suddenly and in successive crops; it is generalized but involves the trunk more than the extremities and consists of numerous dull brownish red acuminate papules or small, dull red nodules which become caseous and break down to form small, shallow round or irregular ulcers which have a thin, purplish red border and a pale, uneven granulating base covered by scanty seropurulent exudate. Miliary tubercles are seen in the base and at the edge of the lesion, and these in turn break down, enlarging the ulcer and causing it to become polycyclic. In rare instances the small, dull red nodules do not ulcerate but undergo spontaneous involution or resorption and disappear. The condition is now very rare, probably owing to the greatly lessened incidence of infection with bovine tuberculosis through milk and the improvement in infant care and child hygiene.

The following report is that of a case of multiple lichenoid papular and small, firm nodular tuberculoderms of the color of normal skin, which ran a noncaseating course over a period of three and one-half years in association with caseous miliary pulmonary tuberculosis. The nodules showed histologic changes typical of tuberculosis with acid-fast rods in the cytoplasm of monocytes and were believed to be blood-borne metastases from the pulmonary tuberculous foci.

REPORT OF CASE

W. L. R., a married white woman, aged 53, was first seen Oct. 28, 1935. There was an eruption of innumerable pale pink, flat nonfollicular papules measuring 2 to 3 mm. in diameter. The papules were discrete but aggregated in large roughly oval areas on the outer surfaces of the arms, the extensor surfaces of the forearms, the anterior surfaces of the thighs and the upper anterior tibial surfaces (fig. 1). There were many small, firm, rounded normal skin-colored nodules measuring 3 to 5 mm. in diameter on the deltoid areas. The eruption was of six months' duration. There was no itching or pain, though slight tenderness of some of the nodules was noted. Scaling, capillary dilatation, erythema and evidence of inflammation were absent. After several months many of the lichenoid papular lesions developed into small normal skin-colored nodules, while others became faint dusky brown and finally disappeared, leaving only pale brownish stains which eventually faded. In places there was some suggestion of grouping. Under pressure with glass a sandy, grayish color clinically suggestive of sarcoid was revealed. There was no evidence of the apple jelly color characteristic of lupus. No lesions were discovered on the scalp, face, neck, trunk, hips, hands or feet or in the mouth. The intervening skin was normal in appearance. New lesions continued to develop, and after the first year the eruption consisted chiefly of small, rounded, scarcely elevated nodules

the color of normal skin, still discrete but more or less closely studded on the extensor surfaces of the arms and legs. All the papular lesions had either developed into small nodules or disappeared, leaving no evidence of atrophy or scarring. After two years, groups of similar small flesh-colored firm nodules developed in

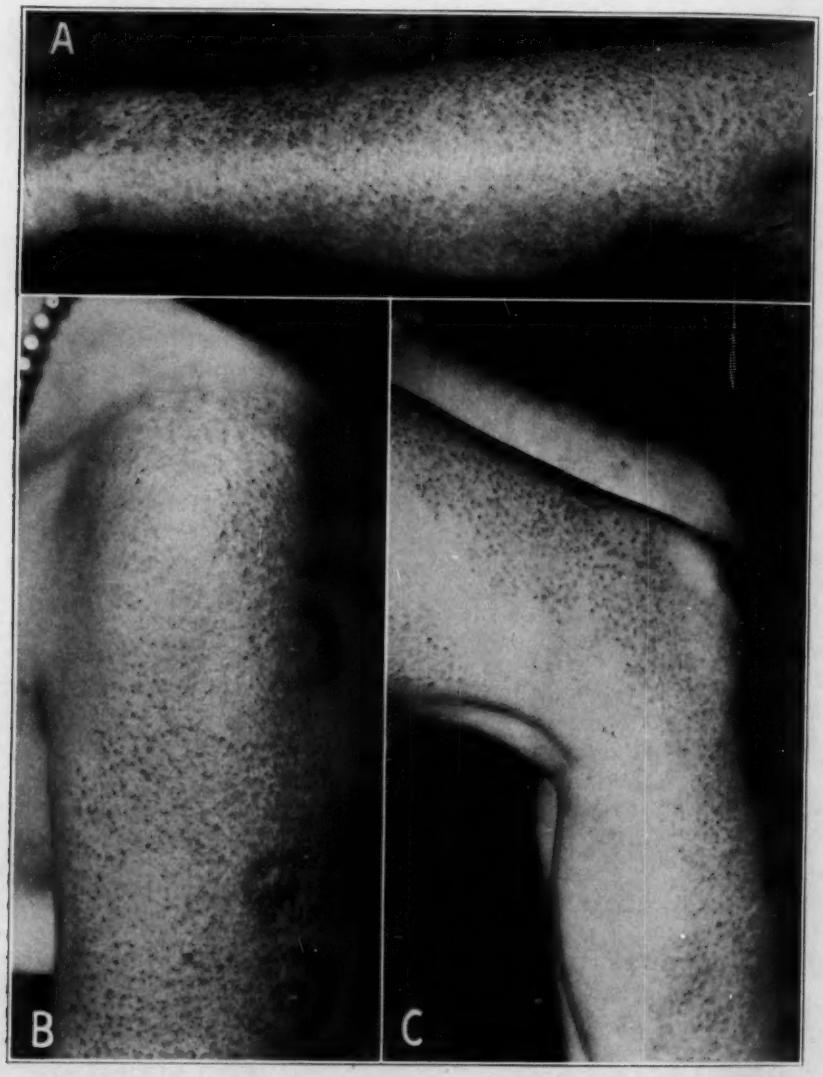

Fig. 1.—*A*, early lichenoid papules on the extensor surface of the forearm. *B*, nodules on the arm. *C*, small discrete colorless nodules on the thigh and leg with one area of grouping.

the suprascapular and infraclavicular regions and over the external surfaces of the thighs. At this time the bridge of the nose showed faint dusky redness and slight roughening suggestive of early lupus erythematosus, which disappeared after several

months. The small nodular eruption continued without change for three years and seemingly disappeared two weeks prior to the patient's death.

Histologic Examination.—Histologic examination of a cutaneous nodule showed typical multiple granulomas, discrete and conglomerate (fig. 2*A*), in the middle and lower layers of the cutis and in the hypoderm. The infiltrate was slightly less dense in the lower layers of the cutis and in the hypoderm. Each small granuloma was of the rosette type and consisted of thirty to fifty central epithelioid cells with no definite necrosis and surrounded by broad mantles of lymphocytes interspersed with a few monocytes. The epithelioid cells were rich in cytoplasm, and the nuclei were large and vesicular, with prominent nucleoli. Some of the central cells had a granular appearance suggestive of early degeneration, but this was confined to one or two cells. A few early giant cells with several centrally clustered nuclei were seen here and there among the epithelioid cells. Grouped acid-fast rods in granular form were found in the cytoplasm of some of the monocytes and early epithelioid cells. Small vascular channels were seen which occasionally showed minute nodular aggregations of three or four cells on the intima; these might possibly have been interpreted as the early intravascular sites of origin of the tubercle before it progressed extravascularly into the cutis. Occasionally a small capillary was seen near the edge of the infiltrate, which showed no evidence of endothelial or medial thickening. The collagen, elastin, follicles, sebaceous glands and sweat glands had disappeared (fig. 2*B*). Acute inflammatory changes and edema were absent. The epidermis was narrowed and covered with a thin layer of keratin, and the rete prolongations had disappeared, leaving the basal line flattened and almost straight. The papillae of the papillary body had likewise disappeared, probably from the pressure of the infiltrate in the underlying cutis. The papillary body was reduced to a narrow strip of loose collagenous fibers sparsely invaded by a few lymphoid cells, and the capillaries showed no dilatation. The normal papillae and rete prolongations reappeared beyond the infiltrated areas.

Past History.—From childhood to the age of 33 the patient had chronic urticaria (wheals), which was often accompanied by throbbing headache over the frontal and temporal regions. Infected tonsils and the appendix were removed in 1922. After a cesarian section in 1925 she had arthritis which lasted several months. She stated that she had repeated night sweats for some time several years before the eruption appeared. There was no history of familial tuberculosis. Her food was a considerable problem for many years because of indigestion after eating; consequently, she ate very little meat, very little starchy or cereal food, few vegetables and no fruits. She was undernourished for several years, which possibly induced decalcification of healed pulmonary tuberculous foci and liberated tubercle bacilli into the blood stream so that the cutaneous lesions developed. She complained of fatigue on slight exertion and of frequent perspiration. There was no cough or expectoration until the fall of 1938, when symptoms of bronchitis developed.

Laboratory Tests.—Repeated examinations of the blood showed average values of 55 per cent hemoglobin, 3,100,000 red cells and 6,400 white cells per cubic millimeter, with 63 per cent neutrophils, 26 per cent small lymphocytes, 7 per cent large lymphocytes and 3 per cent monocytes. The Schilling modification revealed 28 per cent filamented neutrophils, 49 per cent nonfilamented neutrophils, 20 per cent lymphocytes and 3 per cent monocytes. Chemical analysis of the blood showed 11.6 mg. of urea nitrogen, 25 mg. of nonprotein nitrogen, 8.0 mg. of calcium, 3.5 mg. of phosphorus and 425 mg. of sodium chloride per hundred cubic centimeters. The Wassermann reaction of the blood was negative. The urine was normal. Analysis of the gastric contents gave normal results, and examination of a washing

from the stomach failed to reveal tubercle bacilli. Repeated examination of the sputum failed to reveal tubercle bacilli.

Physical Examination.—There were impaired resonance and altered breath sounds at the apex of the right lung. There were no palpable glands in the neck,

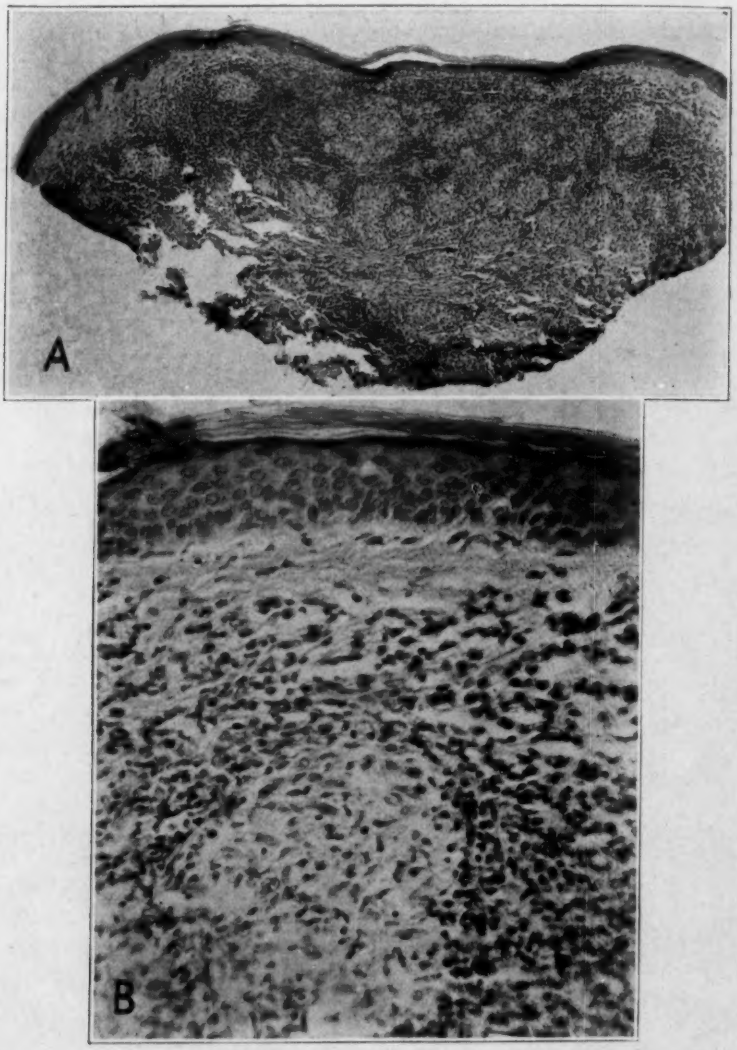

Fig. 2.—Microscopic sections of a cutaneous nodule. *A*, photomicrograph showing numerous discrete and conglomerate miliary tubercles without caseation in the middle and lower layers of the cutis, each surrounded by a wide zone of lymphocytes. The subcutis shows dense lymphocytic infiltrate. *B*, photomicrograph showing the upper layers of the cutis to consist of uninvolved loose collagenous fibers in which the papillae have disappeared together with the rete prolongations, leaving the epidermal line flattened.

axillas or groin. The spleen and liver were not palpable, and there was no abdominal tenderness or palpable mass. The blood pressure was 116 systolic and 90 diastolic; the pulse rate was 85 per minute, and the temperature was 99 F. There were 20 respirations per minute. A roentgenogram of the chest showed a moderate irregular density at the apex of the right lung, extending to the first intercostal space, the greatest density being on a level with the first dorsal vertebra and apparently extending out from the hilus. The left pulmonic field was clear, and the mediastinal space was clear on stereoscopic examination. The Mantoux tuberculin (human) test with a 1:1,000 dilution gave a weakly positive reaction.

Fig. 3.—Microscopic section of the lung, showing a caseous miliary tubercle surrounded by fibrous tissue which has replaced the alveoli and is interspersed with fibroblasts, monocytes, epithelioid cells and lymphocytes. The alveoli shown contain hyperplastic epithelial cells and an occasional polymorphonuclear leukocyte.

Course.—Symptoms of bronchitis developed in the fall of 1938, and a roentgenogram of the lungs revealed a rounded mass extending about 4 cm. from the hilus of the right lung, suggestive of malignant disease. This was revealed by bronchoscopic examination to be located at the junction of the upper and the middle division of the right bronchus. Microscopic examination of a minute biopsy specimen removed through the bronchoscope showed small islands of epithelial hyperplasia suggestive of carcinoma. Total right pneumonectomy was performed

on Nov. 7, 1938, but death ensued on December 8. No metastatic adenopathy was found. Permission for autopsy was not obtained. Gross examination of the right lung showed a miliary inflammatory reaction about the bifurcation of the bronchus, with an area of caseation 3 cm. wide at the base of the upper lobe, near the hilus. There was considerable fibrosis of the upper lobe, extending toward the apex, and also in the upper portion of the lower lobe. Microscopic examination of the lung tissue (fig. 3) revealed miliary tuberculosis with numerous discrete tubercles showing caseation. There was marked fibrosis of the lung tissue with blocking of the bronchioles and alveoli from hypertrophy and hyperplasia of the epithelial lining cells. The tubercles were discrete, showed no evidence of conglomeration and were composed of necrotic caseous centers surrounded by a zone of loosely arranged, roughly radiating epithelioid cells. Each tubercle was surrounded by a loose ring of fibrous connective tissue in which many monocytes, epithelioid cells, lymphocytes and fibrocytes were scattered. Acid-fast rods were observed. A moderate number of multinucleated giant cells of the Langhans type were observed; these were scattered at the periphery of the tubercles among the epithelioid cells and lymphocytes. A few of the tubercles were rather small and showed beginning caseation. There was a decided increase in the interstitial connective tissue, which had scarred over and obliterated large portions of lung tissue. Much of the fibrous tissue was fairly old, with large amounts of collagen separating the flattened fibrocytes. There was diffuse infiltration of chronic inflammatory cells, principally lmyphocytes, with a few plasma cells and an occasional polymorphonuclear leukocyte. The alveoli were partially collapsed, and the lining cells were greatly swollen and somewhat increased in number. The lumen of the bronchi and bronchioles contained some necrotic cell debris mixed with a few red blood cells and occasionally a few macrophages, lymphocytes and (rare) polymorphonuclear leukocytes. An occasional alveolus showed a few lymphocytes and macrophages containing a small amount of pigment. Numerous anthracotic pigment-filled macrophages were seen within the interstitial tissue. There was no evidence of malignant disease.

COMMENT

The eruption is regarded as chronic miliary noncaseating tuberculosis of the skin, blood borne from caseous miliary tuberculous foci of the lungs in an adult with a high degree of resistance. Although the eruption was miliary tuberculosis of the skin, there were none of the features of acute disseminated miliary tuberculosis. The eruption also may be regarded as a cutaneous counterpart of Wende's nodular tuberculosis of the hypoderm, which likewise showed no caseation, though the nodules were larger and evidence of visceral tuberculosis was absent.

SUMMARY

A tuberculoderm is described which began as faint pink lichenoid papules and progressed to small, firm nodules of the color of normal skin. The lesions were discrete yet closely studded and showed some tendency to grouping. Capillary dilatation, inflammation, necrosis and caseation were absent throughout the clinical course of three and one-half years. Some of the lesions disappeared by resorption, leaving no trace except a temporary brownish stain without atrophy or scarring.

Microscopic examination showed each nodule to be composed of discrete and conglomerate tubercles without caseation, each surrounded by a wide zone of lymphocytes interspersed with monocytes and scattered fibroblasts in the middle and lower layers of the cutis. The papillary body was not invaded, but the papillae and the rete prolongations were obliterated from the pressure of the granulomatous process. The subcutis showed dense lymphocytic infiltrate.

The eruption was probably blood borne from foci of caseous miliary tuberculosis of the lungs in a patient with a high degree of resistance.

The eruption is regarded as a small nodular tuberculoderm without caseation, disseminated over the extensor surfaces of the arms and legs.

TREATMENT OF FUNGOUS INFECTIONS WITH ETHYL IODIDE INHALATIONS

A REVIEW

JACOB H. SWARTZ, M.D.
BOSTON

The purpose of this paper is to bring up to date the subject of quantitative inhalation therapy in general, and ethyl iodide inhalation in particular. This paper contains data previously reported plus additional information gathered since the publication of the last paper on this subject. It deals with the following topics:

I. General considerations
II. Chemistry of ethyl iodide
III. Fate of ethyl iodide in the body
  A. Amount of ethyl iodide retained in the body at the end of the inhalation period.
  B. Amount of ethyl iodide exhaled after the end of the inhalation period.
  C. Excretion of iodide in the urine.
IV. Clinical considerations

GENERAL CONSIDERATIONS

Iodide therapy has been used for years in cutaneous and systemic fungous infections. The usual methods of administration have been, first, oral and, later, intravenous. Quantitative inhalation therapy had not been reported previous to the publication by my associates and me of our first paper on ethyl iodide,[1] in February 1930, and no reports of the therapeutic use of ethyl iodide are available in the literature prior to the publication of that paper.

Ethyl iodide was originally chosen because it is an unstable compound which can be absorbed directly into the arterial circulation and

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Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 3, 1939.

1. Swartz, J. H.; Blumgart, H. L., and Altschule, M. D.: Ethyl Iodide Inhalations in the Treatment of Mycotic Infections of the Skin and Allied Conditions, Arch. Dermat. & Syph. **21**:182 (Feb.) 1930.

so reach the peripheral tissues in high concentration. Ethyl iodide is only slightly soluble in water; so intravenous administration is inadvisable. Moreover, if administered intravenously a part of the ethyl iodide would be given off immediately by the lungs before it reached the arterial blood. The investigations of Henderson and Haggard and of Starr and Gamble indicate that more than three quarters of inhaled ethyl iodide is immediately hydrolyzed in the arterial and capillary blood, before it reaches the veins. Some of the ethyl iodide, however, passes into the tissues during inhalation and accumulates there in increasing amounts. This is shown by the fact that after inhalation of ethyl iodide is discontinued venous concentration of the substance exceeds arterial concentration. The exact changes that occur in the peripheral blood are still obscure. Minute amounts of free iodine may conceivably be liberated. The fact that ethyl iodide is a lipoid solvent may also explain in part its therapeutic efficacy. Blood taken from patients before and after the treatment to learn whether the growth of fungi was less in the blood taken after treatment showed no significant difference.

II. CHEMICAL DATA

Ethyl iodide is a monoiodoethane. The formula is CH_3CH_2I; the molecular weight, 155.97; the specific gravity, 1.933; the melting point, — 108.5 C., and the boiling point, 72.2 C. Its solubility in grams per hundred cubic centimeters is as follows:

Water: 0.4% at 20 C.
Alcohol: soluble
Ether: soluble

III. FATE OF ETHYL IODIDE IN THE BODY

A. *Amount of Ethyl Iodide Retained in the Body at the End of the Inhalation Period.*—Studies were carried out as follows: The ethyl iodide was weighed or was measured by means of a United States Bureau of Standards pipet. The desired amount was transferred [2] to a test tube (A, fig. 1).

The apparatus was constructed to permit partial saturation of the inspiratory current of air with ethyl iodide vapor. The two way flutter valve system *B* (fig. 1) communicated directly with a Tissot spirometer of 400 liter capacity in which the total amount of expired air was collected and measured. The inside of the spirometer was coated with

2. Blumgart, H. L.; Gilligan, D., and Swartz, J. H.: The Elimination of Ethyl Iodide After Inhalation and Its Relation to Therapeutic Administration, J. Clin. Investigation **9**:635, 1931.

lead tetroxide (red lead). Samples of gas (250 cc.) were taken from the spirometer and the ethyl iodide content measured according to the method of Starr and Gamble.[3] This method enables one to recover known amounts of ethyl iodide with a high degree of accuracy. The total amount of exhaled iodide could then be calculated. The difference between the amount of ethyl iodide volatilized in the inspired air and the amount exhaled represents the amount remaining within the body at the immediate conclusion of a treatment.

The results of eighteen measurements for 10 different subjects are shown in table 1.[2]

The amount of ethyl iodide inhaled varied from 1.93 to 5.82 Gm. In different persons the percentage of ethyl iodide retained in the body varied from 42.2 to 69 per cent of that inhaled, but in a given subject the percentage retained in successive experiments was remarkably constant regardless of the amount inhaled. In subject S. B., for example, 1.93 to 5.76 Gm. of ethyl iodide was inhaled, but the percentages retained varied only from 42.2 to 46.1. The percentage of ethyl iodide retained

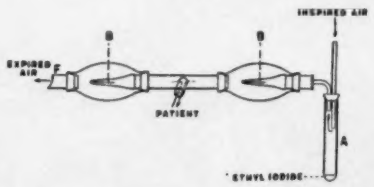

Fig. 1.—Diagram of apparatus used in the investigation. The inspired air is drawn through the test tube (*A*) which contains the ethyl iodide. The concentration of ethyl iodide in the inspired air can be varied by altering the degree to which the straight glass tube projects into the test tube. Rebreathing is prevented by the use of flutter valves (*B B*). The expired air passes through the outlet tube (*F*). For clinical purposes a simpler, more practical apparatus has been devised.

was not affected by the length of time taken for inhalation. It depended to a large extent, however, on the respiratory rate; the lower the respiratory rate, the greater the percentage retention (fig. 2).

With the lower respiratory rate more time is probably available for effective absorption of ethyl iodide by the blood of the alveolar capillaries. In subjects with normal respiratory rates, the average amount of ethyl iodide retained in the body at the conclusion of treatment was 55 per cent of the total amount inhaled.

B. *Amount of Ethyl Iodide Exhaled After End of Inhalation Period.*—Besides the ethyl iodide exhaled during treatment, additional amounts

3. Starr, I., Jr., and Gamble, C. J.: A Method for the Determination of Minute Amounts of Ethyl Iodide in Air, Water, and Blood by Means of Its Reaction with Silver Nitrate and Experiments Bearing on the Determination of Blood Flow by Means of Ethyl Iodide, J. Biol. Chem. **71**:509, 1927.

were exhaled after treatment. To study the latter factor the following methods were used: Ethyl iodide was administered according to the procedure described except that the outlet tube (*F*, fig. 1) was led to the air outside the laboratory to prevent reinhalation of the ethyl iodide exhaled during the treatment. As soon as all the ethyl iodide in the test tube (*A*, fig. 1) had been vaporized, the test tube was removed and the expired air collected in a Tissot spirometer at appropriate intervals for five to ten minute periods. The total volume of expired air during each

TABLE 1.—*Amount of Ethyl Iodide Retained in Body at End of Inhalation Period (from Blumgart, Gilligan and Swartz* [2])

Measurement Number	Subject	Date, 1930	Duration of Inhalation, Minutes	Respiratory Minute Volume, Liters	Respirations per Minute	Amount of Ethyl Iodide Inhaled, Grams	Amount Exhaled During Period of Inhalation, Grams	Amount Retained at End of Treatment, Grams	Per Cent of Total Retained at End of Treatment, per Cent
1	S. B.	January 23	10	10.2	21	1.94	1.12	0.82	42.2
2	S. B.	January 27	26	9.0	23	1.93	1.06	0.87	45.1
3	S. B.	January 30	39	8.5	23	3.64	1.96	1.68	46.1
4	S. B.	February 4	38	8.7	21	5.76	3.14	2.62	45.5
5	M. K.	February 4	35	8.8	30	3.82	2.14	1.68	44.0
6	H. C.	Februarp 6	20	10.6	28	1.94	1.08	0.86	44.3
7	H. C.	February 10	30	11.3	28	3.68	2.08	1.60	43.5
8	H. C.	February 13	33	9.8	25	5.00	2.60	2.40	48.0
9	E. R.	February 3	32	9.0	26	4.96	2.58	2.38	48.0
10	R. A.	January 31	25	11.9	17	4.10	2.00	2.10	51.3
11	J. E.	January 31	40	7.4	13	5.44	2.38	3.06	56.3
12	M. G.	January 24	25	7.2	10	3.88	1.57	2.31	59.6
13	M. G.	January 27	43	7.8	15	3.30	1.40	1.90	57.6
14	H. D.	January 29	26	10.8	17	5.82	2.38	3.44	59.2
15	M. V.	February 14	17	8.0	14	1.94	0.72	1.22	62.9
16	M. V.	February 18	22	6.2	9	1.94	0.73	1.21	62.4
17	M. V.	February 24	47	6.3	9	5.82	2.17	3.65	62.8
18	C. V.	March 14	60	5.5	5	5.82	1.81	4.01	69.0

period was measured, and samples of 250 to 1,000 cc. were taken and analyzed. From these data the total amount of ethyl iodide exhaled during any given period as well as the average number of milligrams of ethyl iodide exhaled per minute could be calculated. By means of these results, curves for excretion [2] were plotted (fig. 3). The final portions of the curves (dotted line) for excretion were estimated, since the concentration of ethyl iodide became too small to be measured.

Indirect evidence in regard to the approximate accuracy of the terminal portions of the curves was obtained by analysis of samples of alveolar air. The concentration of ethyl iodide in the alveolar air is

higher than that of the total expired air, as the expired air collected in the usual manner represents alveolar air diluted by the air of the dead space. The absence of ethyl iodide in the alveolar air collected after the projected portion of the curve had reached zero would be confirmatory evidence of the accuracy of this part of the graph. Ninety to one hundred and twenty minutes after the conclusion of the treatment,

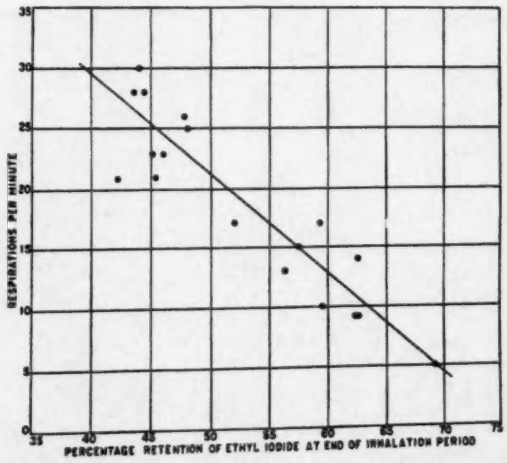

Fig. 2.—Relation between the respiratory rate and the percentage of ethyl iodide retained within the body at the immediate conclusion of the inhalation period.

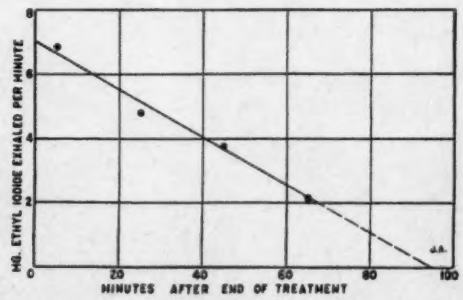

Fig. 3.—Rate at which ethyl iodide is given off in the expired air after treatment.

samples were obtained by collecting the air of successive forced expirations until a total of 1 liter was obtained. Four analyses with 2 subjects showed no detectable amounts of ethyl iodide. The total amount of ethyl iodide exhaled could therefore be calculated by integrating the area under the curve. Thirteen series of measurements were made with 8 subjects. The length of time ethyl iodide continued to be exhaled after

treatment and the total amounts of ethyl iodide exhaled were apparently unrelated to such factors as the dose, the duration of treatment and the respiratory rate of the patient. The average amount of ethyl iodide exhaled after the end of the inhalation period was 0.35 Gm., or 9 per cent of the mean total dose.

C. *Excretion of Iodide in the Urine.*—Other investigators [4] have shown that the major portion of the inorganic iodine salts administered orally, rectally or intravenously is excreted as iodide in the urine. Ten specimens of urine collected immediately after treatment were analyzed for ethyl iodide by the method of Starr and Gamble. No free ethyl iodide could be detected in these specimens. The urine was then collected for ten days after treatment, the time of voiding, the volume and the iodide content of each specimen being recorded. From these data the hourly excretion of iodide was calculated. The amount of iodide excreted in the urine after inhalation of ethyl iodide was compared with that excreted after the administration of similar doses of potassium iodide by mouth. The curves of iodide excretion after administration of ethyl iodide and potassium iodide are of the same general type, the rate and duration of excretion being strikingly similar. Curves showing the percentage excretion during successive twelve hour periods after treatment varied only slightly in all subjects regardless of the dosage. The amount of fluid intake did not alter the duration of excretion, nor did the volume of urine affect the rate of excretion.[2]

Of the total iodide ingested, an average of 75 per cent was excreted by the kidneys. The remainder of the iodide is excreted by the intestines, the skin, and the sputum.

Of the total amount of ethyl iodide not eliminated by the lungs, an average of 68 per cent is excreted by the kidneys as iodide. It is reasonable to conclude that the remainder of the iodide is excreted in the same manner as has been mentioned.

In brief, one may conclude that after 1 to 3 cc. of ethyl iodide is inhaled, an average of 45 per cent is immediately exhaled during treatment, 9 per cent is exhaled during the first two hours after treatment and 31 per cent is excreted by the kidneys. This accounts for 85 per cent of the total dose given, the other 15 per cent being excreted, in all probability, by the intestines, by the skin and in the sputum. Figure 5 illustrates the foregoing remarks.

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4. Cattell, R. B.: The Elimination of the Iodide in the Urine in Normal Persons and in Exophthalmic Goiter, Boston M. & S. J. **195**:69, 1926. Myiri, W.: Kurzfristige, einzeitige Nierenfunktionsprüfung mit Jodnatrium und Thiosulfat, Wien. Arch. f. inn. Med. **9**:511, 1925. Wovschin, W. A.: The Elimination of Potassium Iodid in the Urine: An Experimental Comparative Study of Its Administration by Mouth or Rectum, J. A. M. A. **65**:1101 (Sept. 25) 1915.

IV. CLINICAL CONSIDERATIONS

Ethyl iodide was chosen because of certain advantages which it offered. Ethyl iodide has been used extensively for measurement of the minute volume output of the heart of man [5] and has been found to be nontoxic and highly volatile. The coefficient of distribution between the alveolar air and the blood in the lungs is sufficiently high to allow introduction of a relatively large amount of iodine into the blood stream.

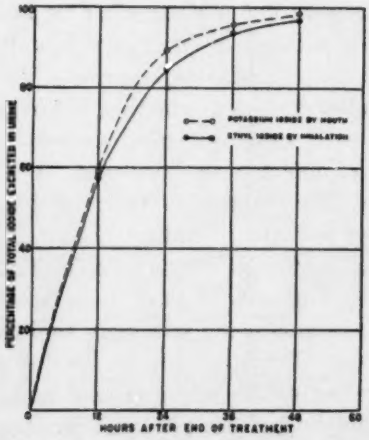

Fig. 4.—Average rate of iodide excretion expressed as percentage of the total excreted in the urine.

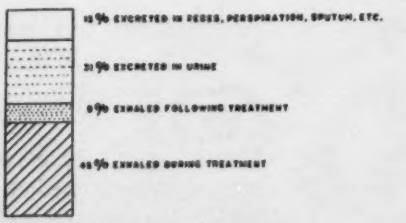

Fig. 5.—Diagrammatic representation of the fate of ethyl iodide originally inhaled.

It has been demonstrated, moreover, that only a small proportion is returned in the venous blood, so that the tissues are exposed to relatively large amounts of iodine.[6]

5. Starr, I., Jr., and Gamble, C. J.: An Improved Method for the Determination of Cardiac Output in Man by Means of Ethyl Iodide, Am. J. Physiol. **87**: 450, 1928.

6. Henderson, Y., and Haggard, H. W.: The Circulation and Its Measurement, Am. J. Physiol. **73**:193, 1925.

It was found that ethyl iodide vapor when not diluted with large quantities of air is unpleasant to the taste and often irritating to the mucous membrane. Accordingly, an apparatus was devised which made possible the administration of any given quantity of the drug with no discomfort to the patient. This apparatus has been modified several times, so that now it is conveniently small, easily manipulated and practical for use at home by the patient. A photograph of this machine has been presented in an earlier publication.[7]

Frequency of Administration and Dosage.—The action of the drug depends to a great extent on its concentration within the body. It is consequently desirable to maintain a high level of concentration of the drug in the body by administering sufficient amounts at appropriate intervals to offset excretion of the substance. Elimination of ethyl iodide takes places through the lungs, the kidneys and the gastrointestinal tract and in the sputum. As previously stated, the results of investigations [2] demonstrated that a total of 54 per cent of inhaled ethyl iodide is exhaled during treatment and within the first few hours immediately following treatment. Measurements of the amounts of iodide eliminated in the urine after inhalation of ethyl iodide offer important evidence regarding the amount remaining within the body, since 68 per cent of the ethyl iodide not exhaled during and immediately after treatment is excreted as iodide in the urine. The rate of excretion is greatest immediately after treatment and decreases rapidly. Approximately 85 per cent of the total urinary iodide is excreted within the first twenty-four hours, and approximately 97 per cent is excreted by the end of forty-eight hours. This indicates that daily repetition of the dose is desirable to maintain a higher level of iodide within the body; that is to say, a given dose repeated daily will maintain a higher level of concentration than twice that dose given every other day. It should be noted, however, that since 15 per cent or more remains within the body, daily repetition will lead to accumulation and ultimately to toxic effects. To prevent such an occurrence, the treatment is omitted every third day, forty-eight hours being allowed to permit almost complete elimination of the accumulated iodide. The amount of the daily dose of ethyl iodide must depend on the therapeutic results and the absence of toxic effects as observed clinically. Originally the dose was 1 cc. (2 Gm.) but since then I have become much more courageous and have increased the dose to as much as 4 cc. (8 Gm.), with better results.

The present regimen is as follows: Begin with 2 cc. (4 Gm.) and and increase 0.5 cc. (1 Gm.) per dose until 4 cc. (8 Gm.) is reached;

7. Swartz, J. H., and Reilly, M.: Inhalations of Ethyl Iodide in Fungous Infections, Arch. Dermat. & Syph. **32**:551 (Oct.) 1935.

continue with 4 cc. (8 Gm.). In systemic fungous infection, doses as high as 5 and 6 cc. (10 and 12 Gm.) have been used without toxic effects. For a child the dose is half that for an adult.

It is important that the room be well ventilated during the period of inhalation. The patient is advised to inhale and exhale through the mouth. The average time required to inhale 2 cc. of ethyl iodide is about twenty minutes. This or larger amounts can be administered in divided doses during the day if the patient is too ill to take it in one sitting. A good time for the inhalation of ethyl iodide is bedtime.

Contraindications to the Use of Ethyl Iodide.—Impaired excretory function of the kidney will probably favor retention of iodide and the appearance of toxic effects. The treatment is therefore contraindicated when such a condition is present. Ethyl iodide should not be administered to patients with symptoms or signs of thyrotoxicosis. It is also contraindicated for patients with active pulmonary tuberculosis.

Results of Treatment with Inhalations of Ethyl Iodide.—Thus far at least 400 patients with various fungous infections of the skin or other organs as well as systemic fungous infections have been treated with ethyl iodide inhalations. The following table is based on the results

TABLE 2.—*Results of Ethyl Iodide Inhalations in 244 Cases*

Condition	Number Treated	Improved or Cured	Unimproved	Percentage Improved	Percentage Cured	Percentage Unimproved
Epidermophytosis	198	168	30	52	32.00	16.00
Cryptococcosis epidermica	20	10	10	35	15.00	50.00
Psoriasis	8	2	6	25	.....	75.00
Tinea capitis	6	4	2	..	66.66	33.33
Chronic paronychia (fungous)	3	1	2	..	33.33	66.66
Favus	5	3	2	..	60.00	40.00
Blastomycosis*	3	3	0	..	100.00	.....
Moniliasis (skin and mucous membranes)	1	0	1	..	.....	100.00

Two patients with extensive involvement caused by Trichophyton rubrum were treated successfully with inhalations of ethyl iodide. These were treated too recently to merit tabulation with those cured, as the term is used in this report.

* In 1 case the diagnosis was made clinically, no mycologic or pathologic studies being made. The number of cases in which blastomycosis was treated is not sufficient to be of statistical value. It simply indicates the advisability of further follow-up with this type of treatment.

observed in 244 cases. The patients were chosen because their records are complete. This table represents the results in terms of percentage cured, improved or unimproved. The term cured was applied to patients who have shown no subjective or objective symptoms for two years or longer. The term improved was applied to patients who were completely freed from subjective or objective symptoms but have shown recurrence within one year, or those of whom follow-up for more than one year was impossible.

The average number of treatments required to obtain cure or improvement was as follows:

Epidermophytosis	27
Cryptococcosis epidermica	24
Psoriasis	40
Tinea capitis	35
Favus	50
Blastomycosis	40

Epidermophytosis involving the hands or feet or both required a greater number of treatments than that involving the folds of the skin. Epidermophytosis with an associated "id" was more responsive to treatment than that without an "id." The results of treatment in a few cases of onychomycosis were so discouraging that no further attempts were made.

The only local treament used was application of a bland cream in cases in which the skin was uncomfortably dry.

In 1 case of bronchomoniliasis treatment with ethyl iodide inhalations was given without recovery, but the organisms failed to be demonstrable shortly before postmortem examination. I believe that if treatment had been resorted to before the patient was close to a moribund condition, recovery would have resulted.

I have received reports of 2 cases of actinomycosis, in 1 of which the kidney was involved, in which the patients were cured with inhalations of ethyl iodide.

Complications.—The most common complication noted was the usual papulopustular eruption, which made its first appearance in the diseased area. The eruption disappeared within a few days after discontinuation of the drug, and we were able to resume treatment in the majority of these cases. In 3 cases peripheral neuritis involving the lower extremities occurred after only a small number of inhalations. The symptoms disappeared in about three weeks after treatment was stopped. Inhalations were not resumed in such cases. This complication was probably due to the ethyl radical.

SUMMARY

A new method of therapy by quantitative inhalation of ethyl iodide is proposed.

The mechanism of action of ethyl iodide is considered.

An improved inhalation apparatus, which is easily manipulated and convenient for use at home by the patient, has been developed.

The optimum dosage and the frequency of treatment, based on the results of investigations to determine the fate of ethyl iodide in the body, are discussed.

The contraindications to use of ethyl iodide are:

1. Impaired excretory function of the kidney.
2. Thyrotoxicosis.
3. Active pulmonary tuberculosis.

The complications following treatment with ethyl iodide inhalations have been chiefly cutaneous eruptions, which made their first appearance in the diseased area, clearing rapidly after treatment was discontinued. Treatment was resumed in the majority of such cases. A more serious complication, peripheral neuritis, occurred in 3 of a series of 244 patients. The symptoms were probably due to the ethyl radical and cleared up in about three weeks after cessation of treatment.

The percentage of patients responding to this form of treatment and the average number of treatments required are presented in tabular form.

Ethyl iodide is not a cure-all for fungous infections, but it is a valuable means of combating the more severe and persistent fungous infections of the skin and mucous membrane. It should be tried for systemic fungous infections and should be of particular value in bronchomoniliasis.

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ABSTRACT OF DISCUSSION

Dr. V. Pardo-Castello, Habana, Cuba: At the time Dr. Swartz presented his first paper on this subject, I happened to have in my ward at the University Hospital a patient with actinomycosis of the lungs with secondary lesions of the skin. I had tried a number of different forms of treatment, with failure after failure. I thought I would try Dr. Swartz's method. I did not have his apparatus, but thought I could use an anesthetic mask, although I would lose some of the ethyl iodide. I do not remember the exact amount of ethyl iodide I used, but the dosage was taken from Dr. Swartz's article. The patient went into deep narcosis immediately after the inhalation, and the narcosis lasted about twenty-five to thirty minutes. On awakening, the patient was irrational and had visual hallucinations. This lasted four days, during which time he had to be restrained. There is a possibility, of course, that the nurse made a mistake and gave a larger amount than was prescribed, but I hardly think so, because I gave her the exact amount to be used out of the ampule in which the ethyl iodide came originally. At the end of four days the patient was quiet, slept profoundly, several hours during the day and at night and finally recovered without any further disturbance. I wonder whether Dr. Swartz has seen any complications in the brain which disturbed the mental condition of this patient. I do not know whether the patient was an alcoholic addict. He finally died of a pulmonary complication.

Dr. Anthony C. Cipollaro, New York: Immediately after the publication of Dr. Swartz's first paper Dr. MacKee and I used ethyl iodide for the treatment of fungous diseases. Over 100 patients with dermatophytosis were treated, and the results were not particularly good. After inhaling from 1 to 2 cc. of ethyl iodide, some patients became groggy. I should like to cite 1 case in order to emphasize the point made by Dr. Swartz, that in certain selected cases ethyl iodide

may be of extreme value. The case was one of generalized psoriasis complicated by moniliasis. The monilial infection involved the skin, mouth, gastrointestinal tract, genitourinary tract and lungs. The patient also had chills and fever of a septic type. Physical signs revealed areas of consolidation resembling pneumonia. After two or three small doses of potassium iodide, severe rhinitis developed. Ethyl iodide in doses of 0.25 cc. three or four times a day was well tolerated, and the patient recovered completely in a short time.

Dr. Louis A. Brunsting, Rochester, Minn.: Perhaps my question has been covered in Dr. Swartz's first article. I should like to ask as to the relative concentration of iodide in the body fluids, particularly in the blood and spinal fluid, obtained with the use of this material as compared with that obtained with the administration of potassium iodide or sodium iodide.

Dr. Jacob Hyams Swartz, Boston: I shall first answer Dr. Pardo-Castello's question. I have anesthetized 2 patients (women) in my office but did not have the experience of their suffering from hallucinations for three or four days. These patients were all right after a half-hour or so and went home and were able to take the treatment again. The reason for the light anesthesia, I believe, was an improper mixture of ethyl iodide and air. That is why I recommend the use of a special apparatus, by means of which one can regulate the amount of ethyl iodide and air. I have given it to children 8 and 9 years old without any trouble. So far as toxic symptoms are concerned, it is important to get pure ethyl iodide, as the presence of phosphorus might cause toxic symptoms. A number of reactions reported to me have been from the use of impure ethyl iodide. I have done some work on ethyl iodide as an anesthetic. I have anesthetized dogs and cats during nephrectomy, following the effect with carotid tracings, and have found that it has a narrower margin of safety than ether, but a smaller amount of ethyl iodide is necessary to anesthetize the animal, and it usually gives a fairly good anesthesia. As far as Dr. Cipollaro's experience with treatment of dermatophytosis is concerned, I will say that in those of the hands and feet it is important to give large doses. The earlier dose recommended does not give as good results as the larger doses I am using now. Those patients with "ids" respond better. I am glad to hear of Dr. Cipollaro's success in his case of moniliasis. I believe that in my case failure resulted from the fact that the use of ethyl iodide was started too late. I cannot answer Dr. Brunsting's question myself but refer him to an article by Blumgart and his co-workers and one by Henderson on the concentration of ethyl iodide in the blood.

GENERALIZED HERPES ZOSTER

REPORT OF NINE CASES

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Herpes zoster is occasionally accompanied by a generalized vesicular eruption, clinically similar to varicella. Some writers believe that herpes zoster and varicella may be caused by different strains of the same virus and that the generalized eruption indicates an infection by both. Others feel that the syndrome is merely a fortuitous concurrence of the two diseases. A third theory, which is based on clinical and experimental observations and seems more probable, is that the generalized eruption is an extension of the zoster itself.

A possible intermediate stage between localized and generalized herpes zoster consists of the appearance of isolated aberrant vesicles, irregularly distributed over the body. These were first reported by Tenneson,[1] who thought they could be found in 9 out of 10 cases of herpes zoster, although other observers consider his estimate too high.

The similarity of these lesions to those of varicella and certain observations as to possible contagion made von Bokay [2] formulate the hypothesis that "the still unknown agent of varicella can, in circumstances which are still unknown, manifest itself not as a general eruption but as a zosteriform eruption. This zoster can later provoke the appearance of typical varicella."

Apart from probable origin in the group of filtrable viruses, herpes zoster and varicella have so many clinical and pathologic differences that a common cause does not appear likely. Nevertheless, until the causative agent of one or both has been isolated, cases of generalized herpes zoster will continue to be of interest.

REPORT OF CASES

CASE 1.—J. B., an Irish laborer aged 74, was admitted to the New York City Hospital with herpes zoster ophthalmicus of three days' duration. Pain developed over the affected area at the time the vesicles appeared and persisted about three

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From the Department of Dermatology of Vanderbilt Clinic and the Columbia University College of Physicians and Surgeons.

Read before the Section on Dermatology and Syphilology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 19, 1939.

1. Tenneson, cited by McEwen, E. L.: The Association of Herpes Zoster and Varicella, Arch. Dermat. & Syph. **2**:205 (Aug.) 1920.

2. von Bokay, cited by Sohn, L.: Zona et varicelle à propos d'un cas d'éruption simultanée, Thesis, Geneva, Switzerland, 1935.

weeks. On the day of admission the patient noticed many blisters scattered over the trunk, extremities and face, accompanied by a slight amount of itching. The zoster lesions were of the severe type, with considerable crusting and secondary infection. The left posterior cervical glands were enlarged and tender. The generalized eruption consisted of many large pinhead-sized to split pea-sized vesicles, most of which contained a turbid fluid. A few lesions were hemorrhagic and some were umbilicated. Most of them were surrounded by an erythematous halo. The temperature was 100 F. on the patient's admission and returned to normal the following morning. The generalized eruption underwent involution within ten days, and the original zoster lesions followed the usual course.

The patient gave no history of chickenpox or of recent exposure to the disease. He had had a syphilitic infection fifty-four years previously, but the Wassermann reaction of the blood had been negative for the past twenty years, and he had received no medication with heavy metal compounds during that time.

Physical examination gave normal results except for the pupils' being irregular, unequal and sluggish. The results of laboratory tests, including Wassermann tests of the blood and spinal fluid, a blood count, chemical analysis of the blood and urinalysis, were normal.

CASE 2.—J. K., a Swedish elevator operator aged 42, was admitted to the Vanderbilt Clinic with herpes zoster on the left side of the thorax of five days' duration. Soreness of the skin developed two days before the eruption appeared, and persisted, accompanied by shooting pains, to the time of the patient's admission. Three days prior to admission a generalized vesicular eruption appeared on the trunk and extremities, consisting of many large pinhead-sized vesicles, most of which were on an erythematous base. Some contained a turbid fluid, and some were umbilicated. Two vesicles were on the mucosa of the lateral wall of the cheek. The generalized eruption underwent involution in about a week, and the zoster lesions healed in about three weeks, with scarring.

The patient had had chickenpox at the age of 5. The results of physical examination and laboratory tests, which included a blood count and chemical analysis of the blood, were normal. An interesting feature was a 4 plus Wassermann reaction of the blood on the day of admission, while four subsequent tests at weekly intervals, as well as a Wassermann reaction of the spinal fluid, were negative.

CASE 3.—N. M., a German laborer aged 36, was admitted to the Vanderbilt Clinic with herpes zoster on the left side of the thorax of three days' duration. The eruption was accompanied by considerable pain, which lasted about ten days. A day after the original lesions appeared an eruption developed on the trunk, scalp and upper parts of the arms and thighs, consisting of many millet seed-sized to split pea-sized vesicles and papulovesicles on an erythematous base. Most of the lesions contained a turbid fluid, and some showed slight umbilication.

The patient gave no history of chickenpox or recent exposure to the disease. The results of general physical examination and laboratory tests, including a Wassermann test of the blood, blood count and chemical analysis of the blood, were normal. A white cell count of 4,800 on the sixth day of the disease may have represented the slight leukopenia which sometimes occurs at that stage.

CASE 4.—J. W., an Irish laborer aged 46, was admitted to the Vanderbilt Clinic with herpes zoster of four days' duration on the left side of the neck accompanied by a moderate amount of pain. Two days before admission a vesicular eruption developed on the trunk and extremities, consisting of thirty to forty large

pinhead-sized vesicles on an erythematous base, some of which were umbilicated. This eruption was accompanied by itching and burning, which disappeared with the involution of the vesicles in about a week.

The results of physical examination and laboratory tests, including a blood count, were normal. The Wassermann reaction of the blood was negative.

CASE 5.—E. S., an American businessman aged 56, a private patient, when first seen had herpes zoster ophthalmicus on the right side of three days' duration, accompanied by a moderate amount of pain and enlargement of the right anterior cervical glands. A vesicular eruption developed on the third day of the zoster, consisting of many split pea-sized vesicles on an erythematous base, some containing turbid fluid. No umbilication was noted. A moderate amount of itching accompanied the generalized eruption, which disappeared in about ten days, and the zoster lesions disappeared in about a month.

The patient had had chickenpox as a child and knew of no recent exposure. The physical examination gave negative results, and the Wassermann reaction of the blood was negative.

CASE 6.—F. G., an American businessman aged 46, a private patient seen through the courtesy of Dr. G. Jarvis Coffin, had herpes zoster of the gangrenous type on the left side of the thorax, accompanied by severe neuralgia, which persisted for nearly four months in spite of various treatments. On the third day a general eruption appeared on the trunk, arms and legs, consisting of large pinhead-sized papulovesicles and vesiculopustules, most of which were on an inflamed base, and a few showed umbilication. The general eruption disappeared in about a week.

The patient had not had chickenpox as a child. Physical examination gave negative results, and the Wassermann reaction of the blood was negative.

CASE 7.—B. H., an American lawyer aged 70, a private patient, had herpes zoster on the lower left side of the abdomen which was preceded by pain and accompanied by general malaise. Three days later several erythematous papular lesions appeared on the face, and a vesicular eruption appeared on the upper parts of the arms, the trunk and the upper parts of the thighs, consisting of pinhead-sized to split pea-sized vesicles and papulovesicles on an erythamotous base. A few of the lesions were umbilicated, and all were painful when pressed. This tenderness and the general malaise disappeared, as if by crisis, on the fourth day of the general eruption, and the lesions underwent involution in about ten days.

The patient had had chickenpox as a child. Three months before the zoster eruption he had had coronary thrombosis and was receiving bromide and digitalis medication. The Wassermann reaction of the blood was negative.

CASE 8.—C. L., a Chinese laundryman aged 40, was admitted to the Institute of Ophthalmology of the Columbia Medical Center with typical herpes zoster ophthalmicus on the left side of six days' duration. Two weeks before he had been struck above the eye and since that time had suffered severe pain in the area. On the fifth day of the zoster an eruption appeared on the trunk and a few scattered lesions on the arms, consisting of pinhead-sized to split pea-sized vesicles, papulovesicles and pustules on an erythematous base. Some of the lesions showed umbilication. The patient had a temperature of 100 F. the first two evenings after admission. The generalized eruption disappeared in ten days, and the zoster lesions were still crusted when he left the hospital two weeks after admission.

There was no history of chickenpox. Physical examination and the laboratory tests, including a blood count and urinalysis, gave normal results. The Wassermann reaction of the blood was negative.

CASE 9.—P. I., a Negro housewife aged 39, was admitted to the Presbyterian Hospital for radiotherapy of mediastinal Hodgkin's disease of ten years' duration, for which she had previously been given extensive roentgen therapy. She was in a debilitated condition and received two transfusions. About twenty-four hours after the second one herpes zoster developed in the right lower part of the abdomen, accompanied by a moderate amount of pain. Five days later a generalized eruption appeared on the face, arms, trunk and upper parts of the thighs, consisting of many small split pea-sized papulovesicles, which, when seen five days later, were crusted, with some hemorrhagic and necrotic.

The patient did not know whether she had had chickenpox as a child and knew of no recent contact. No cases of either chickenpox or herpes zoster had occurred in the ward. The Wassermann reaction of the blood was negative, and the blood count showed 40 per cent hemoglobin, 2,800,000 red cells, 17,900 white cells, 92 per cent polymorphnuclear leukocytes, 6 per cent lymphocytes, 1 per cent mononuclears and 1 per cent basophils.

This patient was seen too recently to be able to report whether any cases arose from contact.

RELATION OF HERPES ZOSTER AND VARICELLA

From the time of von Bokay it has been believed by some that herpes zoster and varicella are two forms of the same disease. Numerous cases have been reported in support of this hypothesis, and McEwen has divided them into four groups: (1) cases in which chickenpox developed after exposure to herpes zoster, (2) cases of local herpes zoster followed by a generalized eruption, (3) cases of generalized herpes zoster followed by varicella in contacts and (4) cases in which herpes zoster developed after exposure to varicella.

Cases [3] falling within the first group are by far the most numerous. Pfeiffer in 1887 was one of the first to remark on the association, and a whole school of authors, headed by Netter in France and LeFeuvre in England, collected cases. Netter himself reported 174 by 1928.

Zoster accompanied by a general eruption, similar to that in each case reported in this paper, seems to have been first described as herpes zoster with varicella by Boullard in 1888, and the literature is con-

3. Cowern, E. W.: Further Observations on the Relationship Between Herpes Zoster and Chicken Pox, Journal-Lancet **56**:590, 1936. Dando, E. A.: Herpes and Varicella, Brit. M. J. **2**:1340, 1913. Goldberg, B., and Francis, F. D.: Herpes Zoster and Chicken Pox, J. A. M. A. **70**:1061 (April 13) 1918. Hombria, R.: Una epidemia de varicella y zoster, Actas dermo-sif. **26**:338, 1934. Le Feuvre, W. P.: "Herpes Zoster" and "Varicella," M. J. South Africa **9**:177, 1914. Netter, H.: Identité d'origine entre un certain nombre de zonas et la varicelle, Thesis, Paris, no. 429, 1921. Orr, J.: Herpes and Varicella, Brit. M. J. **2**:508, 1921. Robson, W. N.: Association of Herpes Zoster and Varicella, ibid. **1**:228, 1921. Stern, E.: The Mechanism of Herpes Zoster and Its Relation to Chicken Pox, Brit. J. Dermat. **49**:263, 1937. Woithelot, G. C. A.: Contribution à l'étude des rapports entre le zona et la varicelle, Thesis, Bordeaux, 1931.

tinually growing.[4] An interesting point to note is the frequency with which herpes zoster, occurring in leukemic patients, is followed by a generalized rash. Craver and Haagensen [4a] stated that it occurred in 50 per cent of their cases.

Reports of chickenpox developing in persons in contact with generalized herpes zoster are rare. Only 7 could be collected from the literature by McEwen [1] and Shelmire and Shelmire.[4h]

The fourth group, consisting of cases in which herpes zoster developed after contact with chickenpox, is represented by less than 20 well authenticated case reports, according to Gunn.[5]

Experimental attempts to demonstrate a common cause have included production of, or immunization against, one form by inoculation with the products of the other, neutralization in animals, cross complement fixation and cross agglutination tests.

Bruusgaard [6] claimed to have produced clinical varicella in a healthy child by inoculation with the contents of a herpes zoster vesicle. Several of the children whom Kundratitz inoculated with herpes zoster and who had positive takes, consisting of a group of umbilicated vesicles on an erythematous base, were subsequently inoculated with the contents of varicella vesicles, with negative results. Chickenpox did not develop in these children even when they were put to bed with patients with active chickenpox or when the mucous membrane of the mouth and nose was smeared with contents of chickenpox vesicles. Kundratitz expressed the belief that he had protected 4 children from a house infection of varicella by injection of convalescent zoster serum.

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4. (*a*) Craver, L. F., and Haagensen, C. D.: A Note on the Occurrence of Herpes Zoster in Hodgkin's Disease, Lymphosarcoma and the Leukemias, Am. J. Cancer **16**:502, 1932. (*b*) Jeanselme, J., and Touraine, A.: Zona suivi d'une éruption généralisée de vesicles abérrantes, Bull. Soc. franç. de dermat. et syph. **25**:351, 1914. (*c*) Kraus, W. M.: The Relation of Herpes Zoster to Chickenpox, New York M. J. **114**:162, 1921. (*d*) Le Févre de Arric and Cohen, C.: Étude expérimentale d'un cas d'herpès généralisé dû à un virus faible, Compt. rend. Soc. de biol. **101**:495, 1929. (*e*) Lynch, F. W.: Cutaneous Lesions Associated with Monocytic Leukemia and Reticuloendotheliosis, Arch. Dermat. & Syph. **34**:775 (Nov.) 1936. (*f*) Minet, J., and Le Clercq, J.: Du zona avec éruption généralisée, Clinique **6**:22, 1911. (*g*) Nisard, J.: Contribution à l'étude pathogénique du zona à propos d'une observation de zona varicelleux chez un pottique, Thesis, Paris, 1930. (*h*) Shelmire, J. B., and Shelmire, B.: Concurrent Herpes Zoster and Varicella, Arch. Dermat. & Syph. **17**:687 (May) 1928. (*i*) Skeer, J.: Herpes Zoster Generalisatus: Report of Two Cases, ibid. **34**:809 (Nov.) 1936. (*j*) Sohn.[2] (*k*) Weber, F. P.: Two Cases of Herpes Zoster Associated with a Generalized Eruption of Varicella-like Spots, Brit. J. Dermat. **28**:13, 1916.

5. Gunn, W.: Chicken Pox, in Rolleston, H.: British Encyclopaedia of Medical Practise, London, Butterworth & Co., Ltd., 1937, vol. 3, pp. 103-115.

6. Bruusgaard, E.: The Mutual Relation Between Zoster and Varicella, Brit. J. Dermat. **44**:1, 1932.

Three attempts were made by Rivers and Eldridge [7] to neutralize the agent of varicella with zoster serum before inoculation into the testicles of monkeys, but only one was successful.

The positive cross complement fixation reactions,[8] using serums and antigens from both diseases, which were obtained by Netter and Urbain, were not confirmed by Lauda and Silberstein, Thomsen or von Bokay, although Brain stated that he had demonstrated them successfully. Glaubersohn and his co-workers had inconclusive results and positive results with serum from patients with syphilis and the same zoster antigen. Agglutination of the elementary bodies of herpes zoster in the presence of varicella convalescent serum has been reported by physicians who believe that these bodies have been conclusively demonstrated.

In order to account for the differing clinical and pathologic pictures of the two diseases, Netter and Urbain developed a theory of mutation of the virus, believing that in varicella it has an affinity for the blood, high infectivity and less resistance than in herpes zoster. In the second form it has, according to the theory, an affinity for the central nervous system and a low infectivity, while its greater resistance would render it capable of provoking an attack after considerable time. This mutation is similar to that which takes place in variola and vaccinia.

The case reports on which so much of the argument for a single agent is based must be subjected to careful scrutiny. After reviewing them, Rivers and Eldridge remarked that most of them were similar to those made a century ago in an attempt to link varicella to varioloid and smallpox and were of questionable value, as varicella is very common and zoster not infrequent. Schamberg suggested that if a relation were postulated between zoster and measles, cases to support the theory could be as readily produced. In reference to the group in which chickenpox causes zoster in contacts, it has been pointed out that since the viruses of both measles and chickenpox are known to localize in areas of irritated skin, some "zoster" which caused varicella could be merely chickenpox with an abnormal localization. Bedson [9] remarked

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7. Rivers, T. M., and Eldridge L. A. Jr.: Relation of Varicella to Herpes Zoster: Clinical and Experimental Observations, J. Exper. Med. **49**:907, 1929.

8. Amies, C. R.: Elementary Bodies of Zoster and Their Serological Relationship to Those of Varicella, Brit. J. Exper. Path. **15**:314, 1934. Brain, R. T.: The Relationship Between the Viruses of Zoster and Varicella as Demonstrated by the Complement-Fixation Reaction, ibid. **14**:67, 1933. Glaubersohn, S. A.; Trachtenburg, S. J., and Blindman, E. O.: Contribution to the Zoster Question, Urol. & Cutan. Rev. **41**:732, 1937. Netter, A., and Urbain, A.: Les relations du zona et de la varicelle: étude sérologique de cent cas de zona, Compt. rend. Soc. de biol. **94**:98, 1926. Netter, A., and Urbain, A.: Le virus varicello-zonateux, Ann. Inst. Pasteur **46**:17, 1931.

9. Bedson, S. P.: Herpes Zoster, Varicella, in Arkwright, J. A.; Bedson, S. P., and others: A System of Bacteriology in Relation to Medicine, Medical Research Council, London, His Majesty's Stationery Office, 1930, vol. 7, pp. 157-168.

on the fact that "of all the various associations of the two diseases which might be expected had they a common etiology, that of chickenpox following zoster within the incubation period of the former disease, alone occurs with sufficient frequency to make chance coincidence improbable." Woithelot stated that in these case reports of contagion the herpes zoster most frequently occurred in adults and the varicella in children.

Bruusgaard [6] suggested that herpes zoster followed by what appeared to be typical varicella in a child might be a case of mixed infection, which is not uncommon in children.

The differing infectivity of the two diseases is another point in favor of a separate causation, since varicella is highly infectious and herpes zoster only slightly so, if at all. In fact, reports of suspected contagion and epidemics are not at all common.[10]

The slight and infrequent rise in temperature at the time the generalized eruption appears is another point of contrast, as adults frequently have considerable febrile reaction in varicella.

It has been demonstrated that herpes zoster, both in its local segmental form and when accompanied by a generalized eruption, presents definite pathologic changes in the central nervous system, occasional lymphocytosis of the cerebrospinal fluid, sometimes motor symptoms and symptoms indicative of involvement of the sympathetic nervous system. This is true in cases of herpes zoster which occur in otherwise normal persons, as well as those which follow infection, medication, intoxication and trauma to the cord, cranial and spinal nerves.[11]

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10. Sainz de Aja, E. A.: Un caso de varicela-zona simultáneos y una pequeña epidemia de zóster, Actas dermo-sif. **25**:246, 1933. Doucet, P. M. C.: Le zona associé aux paralysies et aux amyotrophies, Thesis, Paris, no. 23, 1906. Escaravage, M.: Contribution à l'étude de la contagiosité du zona, Thesis, Paris, 1931. Galippe, J.: Un cas de zona cervical, Bull. Soc. de pédiat. de Paris **15**:200, 1913. Hall, A. J.: Herpes and Varicella, Brit. M. J. **1**:284, 1921. Loireau, J.: Sur une épidémie hospitalière de zonas, Thesis, Paris, no. 301, 1926. Taylor, H.: An Epidemic of Herpes Zoster, Brit. M. J. **2**:13, 1889. Veras, S.: Herpes Zoster by Contagion, Brit. J. Child. Dis. **9**:360, 1912.

11. Batten, F. E.: Acute Anterior Poliomyelitis, Lancet **1**:809, 1916. Baudouin, E.: Les troubles moteurs dans le zona, Thesis, Paris, Troyes, 1920. Bedson.[9] Craver and Haagensen.[4a] Head, H., and Campbell, A. W.: The Pathology of Herpes Zoster and Its Bearing on Sensory Localization, Brain **23**:353, 1900. Hunt, J. R.: The Paralytic Complications of Herpes Zoster of the Cephalic Extremity: A Preliminary Report on the Herpetic Inflammations of the Geniculate, Glossopharyngeal, Vagus and Acoustic Ganglia, J. A. M. A. **53**:1456 (Oct. 30) 1909. Kraus.[4c] Marigonda, P.: Zoster universale emorragico-gangrenoso varicelliforme in individuo con leucemia acuta emictoblastica, Gaz. d. osp. **49**:549, 1928. Marinesco, M. G.: Nouvelles contributions à la pathogénie et à la physiologie du

(Footnote continued on next page)

Varicella, on the other hand, although presenting cutaneous lesions strikingly similar to those of the generalized and localized zoster eruptions and similar inclusion bodies in the epithelial cells, only rarely has complications of the central nervous system.[12] When these occur they take the form of encephalomyelitis, with perivascular demyelination the striking feature of the pathologic picture.

Since recurrence of either herpes zoster or varicella is extremely rare and the diseases are known to establish a lasting immunity, it is interesting to note that reports [13] have been made of herpes zoster in patients who had chickenpox in childhood. Even the idea of an immunity broken after a lapse of time must be questioned, as cases of herpes zoster have been followed in eight or nine months by attacks of varicella.

The tests for cross complement fixation and cross agglutination which apparently gave strongly positive results are open to criticism, since the specificity of the so-called elementary bodies is not proved. Moreover the technic is extremely delicate, and slight errors may give incorrect results. In applying these tests to suspected viruses such errors are prone to occur, as Doerr pointed out. Even conceding the existence of elementary bodies in herpes zoster and varicella infections, which some investigators do not, group agglutination has been demonstrated with dissimilar organisms. Lauda and Silberstein,[14] working along the same lines and using the same technic and virus supplied by the French investigators, were unable to confirm the results Netter and Urbain obtained in cross complement fixation.

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12. Baumgart, H.: Complications nerveuses de la varicelle (les encéphalomyélites post-varicelleuses), Thesis, Paris, L. Rodstein, 1934. Bérode, P.: Les complications nerveuses de la varicelle (à propos des formes méningées), Thesis, Paris, 1932. Boquet, P. A. H.: Les encéphalo-myélities de la varicelle, Thesis, Paris, 1933. Cole, R., and Kuttner, A. S.: The Problem of the Etiology of Herpes Zoster, J. Exper. Med. **2**:799, 1925. Freeman, W.: Neuropathology: The Anatomical Foundation of Nervous Diseases, Philadelphia, W. B. Saunders Company, 1933. Goodpasture, E. W.: Cellular Inclusions and the Etiology of Virus Diseases, Arch. Path. **7**:114 (Jan.) 1929. Rivers, T. M.: Nuclear Inclusions in the Testicles of Monkeys Injected with the Tissue of Human Varicella Lesions, J. Exper. Med. **43**:275, 1926.

13. (*a*) Benad, L.: Contribution à l'étude des rapports entre le zona et la varicelle, Thesis, Paris, 1925. (*b*) Fischer, A. E.: Herpes Zoster and Disseminated Vesicles, J. Pediat. **11**:301, 1933. (*c*) Gay, F. P., and others: Agents of Disease and Host Resistance, Springfield, Ill., Charles C. Thomas, Publisher, 1935.

14. Lauda and Silberstein, cited by Gay and others.[13c]

Studies [15] of the age and seasonal incidence of the two diseases over the same period have been made and have demonstrated that herpes zoster tended to occur in persons over 10 and chickenpox in children under that age. The regular seasonal fluctuations in the incidence of varicella were not followed by herpes zoster, which showed no definite pattern.

In this connection it is also worth mention that zoster has been known to occur in localities in which there was no varicella. Cantor,[16] in his well known report, stated that on Christmas Island, in the Malay Straits, chickenpox was absolutely unknown, whereas zoster was not uncommon. A Norwegian physician, working in an isolated section, reported that in five years he did not see a case of chickenpox, although zoster did occur.

COMMENT

The existence of two types of generalized zoster has been noted. In the first the initial segmental zoster is followed by a succession of typical zonal lesions at the level of different spinal or cranial nerves, which are accompanied by pain and followed by scarring. In the second and more frequent type, a local zoster is followed by a relatively painless scattered erythematous vesicular eruption, without zonal distribution, which disappears without leaving scars. In this group one finds the so-called varicellas, as the eruption bears a striking resemblance to that disease.

Columbini [17] described a case of the first type. The initial eruption involved the three branches of the trigeminal nerve, and the infection spread bilaterally to the territory served by almost all the spinal nerves. Each band of eruption was separated from the next by a band of healthy skin. Bilateral zoster, a more restricted form of this type, also occurred. A possible explanation for these cases is that a severe infection may spread to involve other ganglions. Evidence that such a dissemination of the infection does occur was presented when multiganglionic lesions at different levels were found at autopsy in a case of severe and extensive thoracic zoster.[18]

During the usual course of herpes zoster with vesicular eruption, as reported in the literature under the names of zoster with concurrent varicella, zoster varicellosus and generalized herpes zoster, segmental zoster develops, with outbreak of the generalized phase in a short time. When the local zoster is severe, the general eruption is likely to present

15. Rivers, T. M., and Eldridge, L. A., Jr.: Relation of Varicella to Herpes Zoster: Statistical Observations, J. Exper. Med. **49**:899, 1929.

16. Cantor, S. J.: Herpes and Varicella, Brit. M. J. **1**:227, 1921.

17. Columbini, cited by de Amicis, T.: Caso rarissimo di zoster bilaterale universale, Gior. internaz. d. sc. med. **4**:75, 1882.

18. Levaditi, C.; Lépine, P., and others: Les ultravirus des maladies humaines, Paris, Norbert Maloine, 1938.

similar hemorrhagic and necrotic characteristics. Minet and LeClercq [4f] noted that when the new eruption was separated by any length of time from the original outbreak, it was usually accompanied by the same prodromal symptoms. They further found that the two eruptions did not follow the same course, as the vesicles of the second did not last so long and sometimes did not crust.

Theories as to the method by which the infection in these cases of generalized involvement spreads fall into two groups. According to one, the involvement of one or more posterior nerve roots is followed by spread of the virus, due to an absence of systemic immunity, perhaps through the blood stream into which it passes because of the vascular damage in the ganglion. The second theory is that the attack of the virus on the original ganglion is followed by spread to isolated cells in many other ganglions, thus producing the disseminated vesicles.[19]

The vesicular eruption of both herpes zoster and varicella may be a toxic reaction to the virus, with the virus not present in the vesicles or, if there, destroyed early in the inflammatory process.

It is possible that the agent of varicella is blood borne, and some believe this to be the case. The virus of herpes zoster, however, appears to be one with an affinity for the ectodermal tissues, skin and central nervous system. While it is true that certain viruses may attack both ectodermal and mesodermal tissues, others seem to have a predilection for one system, and the pathologic reports of herpes zoster, in the absence of definite knowledge, tend to support such a hypothesis. Toxic products may, however, pass into the blood stream in severe infections and precipitate the general eruption.

In summary, however, the statement of Parounagian and Goodman [20] in 1923 appears to be borne out by all subsequent investigation: "Lacking bacterial bases for contentions of identity or non-identity of herpes zoster, the generalized eruption sometimes accompanying herpes zoster and varicella, the conclusion to which we must arrive is that there is a clinical group of cases best identified as herpes zoster generalisatus, and that as far as we know now, this is separate and distinct from the fortuitous association of herpes zoster and varicella."

SUMMARY

All the cases here reported occurred in adults. In 3 the local zoster was preceded by pain, and in the others pain developed at the time the lesions appeared. There was general malaise in 1 case, and in 2 the onset of the eruption in its generalized form was marked by fever.

19. Fischer.[13b] Kraus.[4c]

20. Parounagian, M. B., and Goodman, H.: Herpes Zoster Generalisatus: Report of a Case with Review of the Literature, Arch. Dermat. & Syph. **7**:439 (April) 1923.

The zoster in 1 case could possibly be traced to medication and in another to trauma, and it occurred in a third along with Hodgkin's disease. The zoster in the rest was idiopathic. In 2 cases the lesions were of the severe type.

The general eruption developed one to five days after the initial zoster and consisted of pinhead-sized to split pea-sized vesicles on an erythematous base, distributed over the trunk, arms and legs. Some of the lesions became umbilicated, and many contained a turbid fluid. In no case was the general eruption profuse. It was accompanied by slight itching in 1 case; in 2 there was a considerable degree of itching and burning, and in another the lesions were painful when touched. The vesicles did not appear in crops, although in 1 case they spread downward along the trunk and thighs. They underwent involution in a week or ten days, without scarring.

The blood count in 1 case showed slight leukopenia, which may be found in the stage of herpes zoster at which it occurred. Spinal punctures were performed in 2 patients ten days and two weeks after the onset of the zoster, with normal observations.

Two patients gave definite previous histories of chickenpox, and there was no report of varicella in a contact, although the 3 patients hospitalized were in wards. No cases of herpes zoster were reported in contacts.

A positive Wassermann reaction of the blood which was subsequently negative in repeated tests was an interesting feature in 1 case. It seems worth while to note that in another syphilis had been contracted fifty-four years previously but the Wassermann reaction of the blood had been negative the past twenty years.

The similarity of the clinical pictures in the 9 cases observed and the lack of any evidence of varicella, other than the structure of the cutaneous lesions, affords additional evidence that the syndrome is a definite variant of herpes zoster.

CONCLUSIONS

Local herpes zoster may be followed by a general eruption which is due to the same causative agent.

The causative agent may belong to the group of filtrable viruses and may be related to the agent of chickenpox, although it is probably a separate entity.

120 East Seventy-Fifth Street.

ABSTRACT OF DISCUSSION

DR. NELSON PAUL ANDERSON, Los Angeles: Dr. Barker's paper brings up for discussion the whole immunologic mechanism in the diseases caused by virus, particularly those which are so-called dermatropic. Rivers, of the Rockefeller Institute, has arranged these disorders in a table, in this order: sheep pox, horse-

pox, cowpox, smallpox, variola, alastrim, chickenpox, herpes zoster, symptomatic herpes and lethargic encephalitis. As he stated it seems a far cry from sheep pox in the sheep to lethargic encephalitis in man, but when one studies and puts these disorders in a series and then studies their relation, both from a clinical and from an immunologic point of view, there is no doubt that herpes zoster and chickenpox are related. Dr. Barker has ably summed up the evidence for this relation. It may be considered in several ways. The pathologic evidence is not convincing although there are similarities. Acidophilic intranuclear inclusions are present in both conditions.

The same may be said for the immunologic studies which have been done both by complement fixation and by cross agglutination. These tests may simply represent a group reaction which occurs in all of these virus diseases.

Coming to the clinical evidence, there are several types of cases, which, as Barker pointed out, have been more or less systematized by McEwen. First there are cases in which herpes zoster is followed by chickenpox in a susceptible contact. Then one finds the opposite, i. e., cases in which chickenpox is followed by herpes zoster in a susceptible contact. These cases are rare, less than 20 well authenticated examples having been reported. Then there is a miscellaneous group in which zoster has followed zoster, to be in turn followed by chickenpox. Finally, there is the group which Dr. Barker spoke of as cases of herpes zoster generalisatus. This condition has been termed herpes zoster with chickenpox by those who believe that the same virus produces the two conditions. It has also been termed herpes zoster with aberrant vesicles or, as Dr. Barker has called it, a generalized herpes zoster. I do not think a great many cases of this kind are seen, although Dr. Barker has collected a fairly large series. However, I do not believe that they occur with the frequency which some have claimed, namely, that aberrant vesicles appear in practically every case of herpes zoster. I think that most of these cases have occurred in adults. In some of them there was a history of chickenpox in childhood, as in 3 of Dr. Barker's 9 cases. Only 1 case has been reported in which the chickenpox-like eruption has appeared first and has been followed later by herpes zoster. This, however, might be a zosteriform appearance of chickenpox.

I believe that Dr. Barker's first explanation as to the occurrence of such a generalized eruption was probably correct, namely, that the virus gains access into the posterior root ganglion and later spreads into the general circulation and produces a generalized eruption. I think that many of the patients in whom we see a severe generalized eruption have herpes zoster of severe, and many times of hemorrhagic, nature.

I agree with the first conclusion, namely, that local herpes zoster may be followed by chickenpox or by a generalized eruption and that these two conditions are due to the same etiologic agent.

The second conclusion, that this etiologic agent is probably not identical with that of chickenpox, I think must for the time being be considered undetermined. The excellent clinical experiments of Bruusgaard particularly and also of Kandratitz are conclusive evidence that the virus producing these two diseases, if not identical, at least is as closely related as the viruses which produce smallpox and vaccinia.

DR. JOSEPH GRINDON JR., St. Louis: I had the good fortune of observing, within one week of the past year, 2 cases of herpes zoster distributed along the trigeminal nerve and accompanied with a generalized eruption. Reports of these cases were published in the May issue of the ARCHIVES (ARCH. DERMAT. & SYPH. **39**:865, 1939).

The first case was that of a white man over 60 who presented extensive zoster distributed along the right trigeminal nerve. Two days after the onset of the facial zoster, a generalized eruption resembling varicella was observed.

The second case was also that of a white man over 60 in whom herpes zoster was distributed along the right trigeminal nerve. Also vesicles were grouped on the right side of the neck, extending to the level of the third cervical vertebra. Four days after the zoster was noted a generalized varicelliform eruption was observed. Ten days after the onset of the zoster paralysis of the right facial nerve developed.

The generalized eruption in both cases consisted of small, friable vesicles surmounting erythematous bases. In both cases all lesions appeared at one time, that is, there were not crops of lesions. The absence of crops distinguished these eruptions from varicella.

With Dr. Barker, I think that because the generalized eruption accompanying herpes zoster has no crops, there may be accompanying involvement of the motor nerves, and the fact that some of the patients give a definite history of varicella indicates that this generalized eruption is not varicella but in all probability is dependent on the dissemination of the virus of herpes zoster.

Dr. Leslie P. Barker, New York: Dr. Grindon's 2 cases offer additional evidence that generalized herpes zoster occurs most frequently in adults. In my series, all patients were over 40.

The generalized eruption appears from one to six days after the initial zoster lesions.

Suggestive evidence of toxemia or infection was afforded by the fact that in 1 case in which there was general malaise and pressure pain in the lesions of the generalized eruption, the symptoms disappeared as if by crisis on the fourth day.

Although the field of viruses is as yet little understood, in the case of herpes zoster the generalized outbreak appears to have the same cause as the local eruption.

A FAMILIAL PIGMENTARY ANOMALY

S. WILLIAM BECKER, M.D.
CHICAGO
AND
MAURICE J. REUTER, M.D.
MILWAUKEE

Before the advent of modern study of pigment,[1] in which the dopa reaction of Bloch and the silver nitrate reaction for darkening melanin are the most essential methods, it was difficult to diagnose many dermatoses characterized predominantly by abnormality of melanotic pigmentation. Since that time many conditions have been more or less satisfactorily evaluated as far as the actual pigmentary process is concerned. The underlying etiologic mechanism may, however, defy all efforts at solution. There are still unusual pigmentary dermatoses which for the very reason of their rarity have not been observed frequently enough to establish their nature. It is only after such a condition has been reported by several observers that it is eventually pigeonholed for future reference. Such a condition was recently studied by us, which, owing to its rarity and interesting features, was deemed worthy of report.

REPORT OF THREE CASES

Three sisters, 1 aged 47 and the other 2 twins aged 54, had had pigmented macules on the chin, neck and arms as long as they could remember. The lesions were brown, varied from 1 mm. to 1 cm. in diameter and where closely set became confluent to form a pigmented network, with normal-appearing skin in the interstices. All 3 patients stated that the lesions became darker when they were nervously excited. This effect was transitory and lasted only a short time. A total hysterectomy had been performed on the eldest sister at the age of 31, after which, she believed, the lesions had become more pronounced. The father was living and well at the age of 91 and had a similar pigmentary disorder. His sister died at the age of 86 and also had the condition.

The patients stated that exposure to sunshine during the summer produced tanning between the lesions but little or no change in the lesions themselves. Exposure of the normal skin on other parts of the body resulted in diffuse tanning. Hypersensitiveness to the sun's rays was not definitely present in any of the three,

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Read at the Sixty-Second Annual Meeting of the American Dermatological Association, Inc., Monte-Bello, Quebec, Canada, June 1, 1939.

From the Section on Dermatology of the Department of Medicine of the University of Chicago and from the private practice of Dr. Reuter.

1. Becker, S. W.: Melanin Pigmentation, Arch. Dermat. & Syph. **16**:259 (Sept.) 1927.

although the twins complained of burning and itching of the involved regions in the summer. None of them had suffered from photophobia, one of the cardinal symptoms of xeroderma pigmentosum, or had ever noticed erythema, scaling or crusting over any part of the body.

Physical examination revealed all 3 patients to be healthy. The lesions on the neck and on the forearms faded out gradually into the normal areas (figs. 1 and 2). Although the skin appeared slightly thin, there was no definite loss of elasticity as seen in senile atrophy of the simple or the degenerative type. There were no atrophic macules or telangiectases.

Microscopic Examination.—Two sections were removed from pigmented macules on the neck, one for routine staining and the other for the dopa reaction. Routine fixation with Bouin's solution and staining with hemalum, erythrosin and saffron (fig. 3) showed the stratum corneum to be thickened and loose. The inner portion of the stratum corneum, however, was dense and firmly adherent. The stratum granulosum consisted of one layer of cells and was fairly uniform throughout the center of the section. Toward the end of the section, where the epidermis was more nearly normal, it consisted of two or three layers of cells. The stratum mucosum was either aplastic or atrophic, varied in thickness and in its thinner portion consisted of only two layers of cells. Normal papillae were for the most part absent, and the rete processes extended irregularly into the cutis, united with each other in some places and usually had clubbed inner extremities. In some areas these clubbed ends had been cut off and appeared as islands of epithelial tissue. There was little tendency to palisade arrangement of the basal cells except at the ends of the section, where the epithelium was more nearly normal. The cutis was involved in mild round cell perivascular infiltration.

A preparation with silver nitrate (fig. 4) showed the pigment to be melanin. It was located almost exclusively in the basal portion of the epidermis, especially at the proximal ends of the rete processes. There were few chromatophores, which is in accordance with the absence of a grayish hue of the lesions. Only a few melanoblasts could be identified, and none of them were of the dendritic type, showing that the pigmentary process was not particularly active at the time the section was removed.

The dopa reaction showed only an occasional positive cell in the basal layer, with no branching. This may be interpreted as a pigmentary process quiescent at the time the specimen was removed.

DIFFERENTIAL DIAGNOSIS

Failure to discover more than the normal number of pigment-forming cells classifies the condition as melanosis not associated with an increased number of melanoblasts. This eliminates ephelides, pigmented macules in von Recklinghausen's multiple neurofibromatosis, pigmented nevi and neoplastic lesions. Melanosis associated with a normal number of melanoblasts [2] may be due to external or internal causes or to a combination of the two. Among the external causes of melanosis are various physical agents, such as ultraviolet rays, roentgent rays, radium rays and alpha rays from thorium x. Actinic rays

2. Becker, S. W., and Obermayer, M. E.: Modern Dermatology and Syphilology, Philadelphia, J. B. Lippincott Company, to be published.

Fig. 1.—Pigmented lesions on the neck, arms and forearms in 3 sisters.

Fig. 2.—Pigmented lesions on the neck.

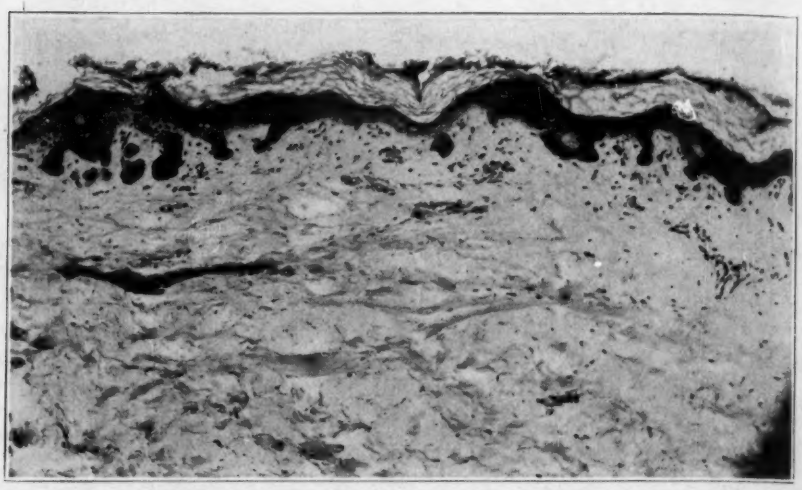

Fig. 3.—Low power photomicrograph of a section from a lesion on the neck. Hemalum, erythrosin and saffron stain.

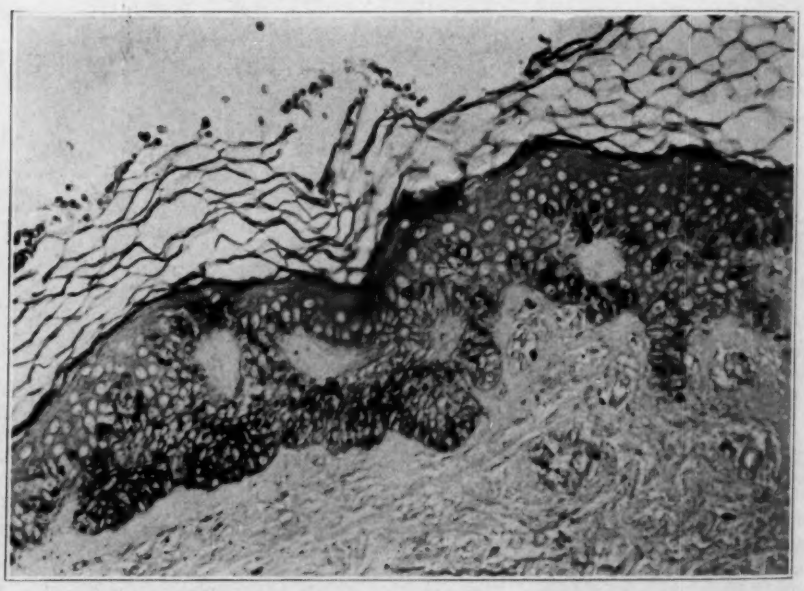

Fig. 4.—High power photomicrograph of a section from a lesion on the neck, showing the reaction to silver nitrate.

may act by virtue of their pigment-stimulating properties only, or the action may be enhanced by sensitization of the skin either by substances applied externally, such as tars, or substances ingested or injected, such as the various acridin derivatives, especially acriflavine (trypaflavin). The resulting melanosis is characterized by brownish pigmentation diffusely distributed over the exposed region. When an element of sensitization is present, more intense and more persistent pigmentation results, with a grayish tint due to deposition of melanin in chromatophores in the superficial layers of the cutis.

Melanosis from heat rays (melanosis calorica) is characterized by retiform brownish pigmentation with much coarser network than is observed in the condition we are reporting. It is confined to regions which have been exposed to radiant heat (as in stokers), to conduction of heat (as in the case of a hot water bottle or an electric pad) or to convection of heat. Mechanical irritation, such as pressure and friction from a truss pad, results in diffuse brownish pigmentation. Accentuation of melanosis due to friction and pressure is noted in patients with Addison's disease. In vagabondism, in which there is an element of mechanical irritation, the involved region, usually the upper part of the back and the region about the waist line, is grayish brown with numerous linear excoriations.

Chemical irritants, especially oils and tars, may produce melanosis with grayish brown tinting on account of pigment in chromatophores. Chemical melanosis has been seen, particularly during the war. Oppenheim attributed the condition to the poor quality of the machine oils and tars and to lack of appropriate hygienic measures. While this dermatosis was predominantly inflammatory and pigmentary, other changes, such as hyperkeratosis, comedo formation, folliculitis, acne vulgaris and at times carcinomatous degeneration, occurred. In the origin of "war melanosis" endogenous factors, such as vitamin deficiency and adulteration of foods with liquid petrolatum, probably also played some role. This emphasizes a close relation to Riehl's melanosis,[3] 18 cases of which were reported in Austria. No new cases have been observed since 1918, and Kumer stated the belief that subsequent reports do not conform to Riehl's original description. The melanosis was attributed to the ingestion of substitute material used in Austria for making bread. Sunlight undoubtedly played a role. The onset was gradual and predominantly pigmentary; inflammatory phenomena were almost completely absent; the scalp was involved, and the condition persisted indefinitely. The patients have been observed periodically,

3. Kerl, W.: Ueber die "Melanose (Riehl)," Arch. f. Dermat. u. Syph. **130**: 436, 1921.

and in none of them has the melanosis completely disappeared. Civatte [4] described a similar condition under the name of poikiloderma, which is now usually designated poikiloderma (Civatte) to distinguish it from poikiloderma (Jacobi).

Among the various internal causes of melanosis, ingestion of arsenic was formerly a common offender. The resulting pigmentation is diffuse over most of the body, especially the trunk; it is brownish or slightly grayish brown, small areas being free from pigmentation. Palmar and plantar keratoses may accompany this eruption. Erythrose peribuccale pigmentaire (Brocq) [5] represents a combination of melanotic pigmentation and erythema, with the latter predominating. The lesions consist of brownish plaques about the mouth, which vary in intensity from time to time. Microscopic examination shows active formation of pigment with more definite changes in the underlying vascular bed. The accentuation of lesions in our patients due to nervous excitement suggests the same combination of vascular and melanotic change as is seen in erythrose peribuccale pigmentaire, with melanotic change predominating.

Melanoses of internal causation belong to the postinflammatory group. Chronic dermatitis, dermatitis herpetiformis, urticaria followed by pigmentation and urticaria pigmentosa are examples. The pigmentary phase of xeroderma pigmentosum represents a response to ultraviolet light in a hypersensitive skin. The clinical lesions are evident on the face, hands and arms. The initial change is intense erythema following exposure to the sun. This is followed by pigmentary macules, scaling, keratotic lesions and, later, telangiectasis and atrophy. Various benign and malignant tumors are also seen. Becker, Pautrier and Woringer [6] found the dopa reaction to be intermittent, being most marked in regions with the greatest amount of dermal pigment. In sections treated by the silver nitrate method melanin was present intermittently in large quantity. The most inflamed regions generally contained less, but other regions also were deprived of pigment. In the pigmented area most of the melanin was in the basal layer of the epidermis and in the adjacent layers. In the intensely pigmented regions numerous dendritic melanoblasts were seen.

Generalized scleroderma is associated with hyperpigmentation and hypopigmentation. In Addison's disease there is slowly progressing

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4. Civatte, A.: La poikilodermie reticulée pigmentaire du visage et du cou, Presse méd. **30**:572, 1922.

5. Obermayer, M. E., and Becker, S. W.: Erythrose Peribuccale Pigmentaire of Brocq, Arch. Dermat. & Syph. **26**:444 (Sept.) 1932.

6. Becker, S. W.; Pautrier, L. M., and Woringer, F.: A Case of Xeroderma Pigmentosum, Arch. Dermat. & Syph. **25**:915 (May) 1932.

hyperpigmentation, more severe on portions of the body normally hyperpigmented. The skin becomes a diffuse brown, and the hair has a brownish sheen. Melanosis accompanying pregnancy may be designated as physiologic. Certain portions of the body are hyperpigmented, namely, the nipples and areolas, the axillas, the genitalia and the midline of the abdomen (linea nigra). This pigment persists to a certain extent after delivery. Chloasma is a melanotic disease the cause of which has not been satisfactorily determined. The lesions are located on various parts of the body, especially on the face, and consist of large brown macules which darken on exposure to ultraviolet light. The condition is occasionally associated with pelvic disturbances, including pregnancy (chloasma uterinum).

Lentigo designates a macular lesion, usually 0.5 to 1 cm. in diameter. It is brown and is seen predominantly on the backs of the hands and on the face. It ordinarily appears late in life but may have its onset at an earlier date. On microscopic examination pigmentary activity is seen which resembles that occurring after external exposure to ultraviolet light. In certain instances multiplication of melanoblasts takes place, constituting lentigo maligna, precursor of clinical malignant melanoma. In other instances a microscopic picture is seen which resembles that in the cases we are presenting, to be mentioned shortly. Acanthosis nigricans consists of lesions at first only slightly elevated and velvety but later rugous and brownish or grayish brown, located in the axillas, in the inguinal region and about the neck.

From a perusal of the foregoing paragraphs it is seen that the dermatosis observed in our 3 patients does not correspond clinically or microscopically to any of the more common pigmentary dermatoses characterized by a normal number of melanoblasts. Nor does it correspond to more unusual types of pigmentary disturbances, such as the generalized hyperpigmentation reported by Wende and Baukus,[7] which from the description resembled Riehl's melanosis, or pigmentary disturbances associated with cutaneous dystrophic changes, such as those reported by Engman[8] and by Cole, Rauschkolb and Toomey.[9] It is, of course, possible and even probable that a pigmentary dermatosis similar to that in our patients has been recorded, but we have been

7. Wende, G. W., and Baukus, H. H.: A Hitherto Undescribed Generalized Pigmentation of the Skin Appearing in Infancy in Brother and Sister, J. Cutan. Dis. **37**:685 (Oct.) 1919.

8. Engman, M. F., Jr.: Congenital Atrophy of the Skin, with Reticular Pigmentation, J. A. M. A. **105**:1252 (Oct. 19) 1935.

9. Cole, H. N.; Rauschkolb, J. E., and Toomey, J.: Dyskeratosis Congenita with Pigmentation, Dystrophia Unguis and Leukokeratosis Oris, Arch. Dermat. & Syph. **21**:71 (Jan.) 1930.

unable to find the reports in the literature. It is possible also that such cases are not as rare as it would seem and that owing to the harmlessness of the dermatosis it has been disregarded. For instance, in discussing Engman's case of reticular pigmentation[10] presented at the Chicago Dermatological Society, Pusey stated that he had observed a patient for several years with a pigmented area around the neck with punctate white mottling, which in his opinion was of prenatal origin.

The only instance we have been able to find of dermatosis closely resembling or actually identical to that of our patient is that reported by one of us (M. J. R.) in collaboration with Montgomery.[11] A woman aged 42 had had pigmented macular lesions on the arms and neck since the age of 17. There was no other instance in the family. The lesions were brownish black, were located on the neck and forearms and varied from a few millimeters to 1 or 2 cm. in diameter. Interspersed with the pigmented macules were erythematous macules containing telangiectasis. The lesions were less prominent over the face and forehead. Flesh-colored papules were seen on the abdomen and thorax. Microscopic section showed findings similar to those in our cases. A diagnosis of xeroderma pigmentosum was made, largely from the similarity of the microscopic section to an illustration so labeled in Gans' "Histologie der Hautkrankheiten."[12] In considering this diagnosis in our cases, we noted that this microscopic picture did not conform to that reported by a majority of authors who had studied the microscopic picture in xeroderma pigmentosum, including one of us (S. W. B.),[6] but may represent a less common variety. A personal communication from Gans stated the conviction that the section was removed from a patient with the condition who also had a carcinoma of the nose and that this microscopic picture represents a certain stage of the disease.

In searching further for similar microscopic changes, we noted an illustration presented by Miescher,[13] labeled "lentigo of the dorsal surface of the hand." He stated that the significance of these melanotic lesions in old persons is not yet clarified. One of us (S. W. B.) has seen 2 patients whose lesions showed a similar microscopic picture. The first was a woman aged 60 who had had a lesion on the dorsal surface of the left hand for several years. Itching and pain had recently

10. Pusey, in discussion on Engman: A Unique Case of Reticular Pigmentation of the Skin with Atrophy, Arch. Dermat. & Syph. **13**:685 (May) 1926.

11. Montgomery, H., and Reuter, M. J.: Xeroderma Pigmentosum, Arch. Dermat. & Syph. **26**:256 (Aug.) 1932.

12. Gans, O.: Histologie der Hautkrankheiten, Berlin, Julius Springer, 1925.

13. Miescher, G.: Melanom, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1933, vol. 12, pt. 3.

developed. Her father had had carcinoma of the skin. Section (fig. 5) showed the stratum to be hyperkeratotic. The stratum granulosum varied from one to several cells in thickness in different parts of the section. The epidermis over the papillae was thin, and the rete processes were long and often joined at their proximal portions with their neighbors on either side. The rete processes often consisted of only two layers of cells, and they seldom had a definite palisade arrangement. The superficial layers of the cutis contained several rather large collections of loosely arranged lymphocytes. The silver nitrate reaction (fig. 6) showed the melanin predominantly in the basal portion of the epidermis, and considerable pigment was seen even in the stratum corneum. No dendritic cells could be seen in the epidermis, and the superficial layers of the cutis contained a large number of chromatophores filled with melanin. A diagnosis of lentigo or an abortive type of verruca senilis (epithelioma) was made.

Fig. 5.—Low power photomicrograph of a section from a pigmented lesion on the back of the hand. Hemalum, erythrosin and saffron stain.

The second patient was a woman aged 54 who had noted for five or six years several small, firm, slightly depressed lesions with what she called "shrinking of the skin" over them. They were located on the arm, leg and buttock. They were flesh colored and slightly depressed, measuring 0.5 to 1 cm. in diameter. Clinically they were thought to represent subepidermal fibrosis. Microscopic section (fig. 7) showed the epidermis to be normal at one end of the section except for slight hyperkeratosis. Toward the center the epidermis was thinned at the tips of the papillae, but the rete processes were elongated. These processes were often rather thin but at times were thickened and in

many instances were united with adjacent processes by means of bars of cells with hyperchromatic nuclei extending across at their inner extremities. The cutis was more cellular than normal and evidently somewhat thinned. The collagen appeared in the form of small strands

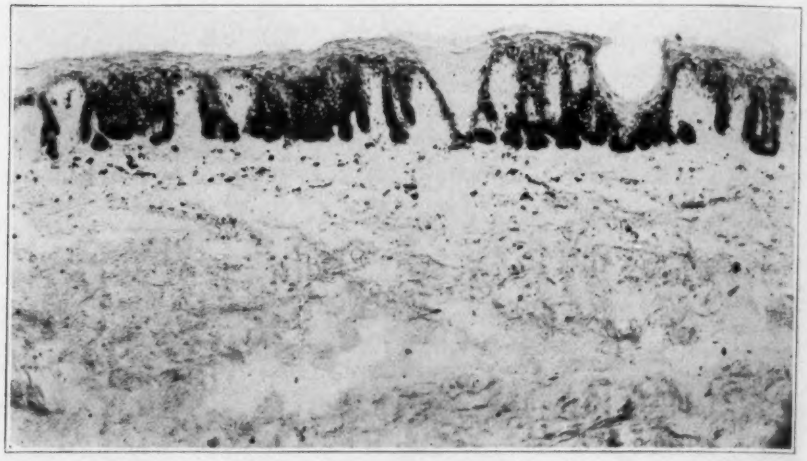

Fig. 6.—Section from the lesion in figure 5, showing the reaction to silver nitrate.

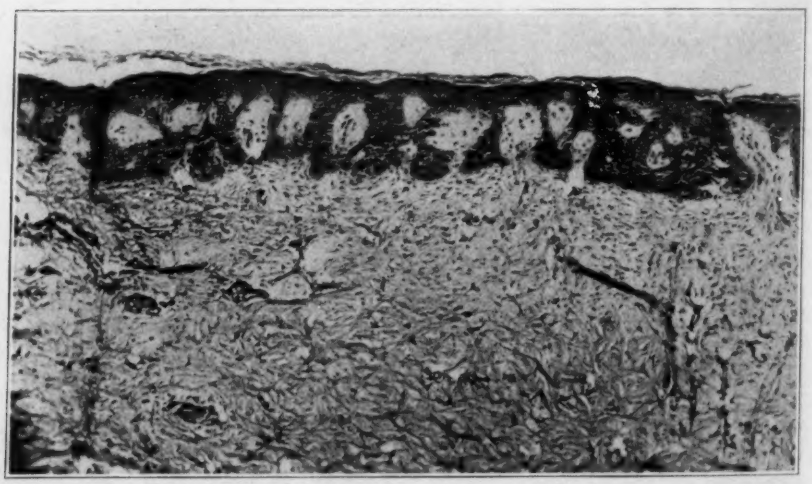

Fig. 7.—Low power photomicrograph of a section from a lesion on the thigh. Hemalum, erythrosin and saffron stain.

that coursed in many directions rather than in the normal large bundles. A preparation with silver nitrate showed only a small amount of melanin, chiefly in the epithelium along the outer portion of the papil-

lae. Microscopic examination thus verified the clinical impression of fibrosis, but the epithelial change is difficult to explain. It may represent an abortive type of senile verruca.

In consideration of the possible diagnosis of xeroderma pigmentosum, in which Gans has found a similar microscopic picture, both our patients and those of Montgomery and Reuter lacked the essential prerequisite for the diagnosis, namely, photosensitiveness. The burning and itching of the involved regions in the summer in 2 of our patients suggest the possible influence of sunshine. The appearance of the pigmentary dermatosis in 3 sisters, the father and an aunt does not conform with the recessive inheritance attributed to the disease by Siemens.[14] Differentiation from the other pigmentary diseases enumerated presents little or no difficulty. We are therefore compelled to conclude that the condition in our 3 patients represents a unique clinical entity which has not previously been identified and which does not conform to the picture of any of the common or unusual pigmentary dermatoses.

Examination of about 500 specimens yearly has shown this unique microscopic picture only 3 times in twelve years of clinical practice. The fact that the 2 isolated instances and Miescher's illustration are all of lesions in elderly persons suggests that the epidermal change is an involutionary one. It must be noted however, that no other signs of simple or degenerative senile atrophy [2] were clinically visible in the 3 patients. Pigmentary lesions (lentigines) are a part of degenerative rather than of simple senile atrophy. The lesions in our patients were on the neck and arms, two regions ordinarily involved in degenerative senile atrophy, but freedom of the face, which is the most common site for the condition, suggests that if the change was an involutionary one it was unique in that it occurred alone. Since the condition occurred at an early age, it is impossible to state with certainty whether the thinning of the epidermis represents aplasia or atrophy. The overgrowth of the rete processes represents some form of dysplasia.

SUMMARY

Three cases of a unique pigmentary dermatosis in sisters, 2 of whom were twins, are reported. The lesions consisted of discrete and confluent brown macules varying from a few millimeters to 1 or 2 cm. in diameter, located on the neck and forearms.

Microscopic section showed epidermal aplasia or atrophy over the papillae, with dysplasia of the rete processes. The silver method showed

14. Siemens, H. W.: Die Vererbung in der Aetiologie der Hautkrankheiten, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1929, vol. 3.

the process to be rather inactive, since only a normal number of nondendritic melanoblasts were present. This was verified by the dopa reaction.

The condition may be classed with melanoses due to hyperactivity of a normal number of melanoblasts. The condition does not conform to any of the common or uncommon pigmentary dermatoses belonging in this group.

It is hoped that this brief discussion will encourage the reporting of others cases of this kind.

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ABSTRACT OF DISCUSSION

Dr. Harold N. Cole, Cleveland: As I looked at the slides of these sisters showing this peculiar discrete pigmentary process on the neck, I felt that the lesion resembled that of a patient my associates and I presented in Cleveland. Many of the members here have seen this patient. The condition was also like that described by Dr. Engman, whose patient I have not seen personally. The condition in the cases reported by Dr. Becker and Dr. Reuter is likewise a familial type of disease. It is true that there are no changes in the nails, hair or mucous membranes. That is possible, however, as there are all types and gradations of the disease. The condition in the case referred to by me probably falls in the same category. The name that Jadassohn gave to the condition was pachyonychia congenita, a designation for the changes in the nails only. We suggested the name dyskeratosis congenita with pigmentation, dystrophia of the nails and leukokeratosis oris (Cole, H. N.; Rauschkolb, J. E., and Toomey, J.: Dyskeratosis Congenita with Pigmentation Dystrophia Unguis and Leukokeratosis Oris, Arch. Dermat. & Syph. **21**:71-93 [Jan.] 1930). This designation at least is a little more descriptive.

Dr. Samuel W. Becker, Chicago: I appreciate Dr. Cole's discussion, because I did not see the patient mentioned by him but only had the opportunity of reading the description of the case. It may be that we shall have to call this the forme fruste of the disease described by Dr. Cole and his associates. I feel, however, that these cases are not as unusual as it would seem. The same applies to pseudoatrophoderma colli, which Dr. Frost is going to discuss. When one reports a case, it is surprising how many others turn up. That was our reason for reporting these. There are probably other cases of lesions similar to the condition described by us, called freckle or lentigo, and we felt it desirable to call attention to it so that other cases may be recorded.

Note.—Since the paper was written additional family history has been obtained which was not available previously. Our 3 patients came from a family of 16 children. Of these, 2 died early in life. None of the 10 had pigmentary lesions. Of the 6 living children, 1 brother, aged 43, also has the anomaly. The father, who also had the lesions, had 4 other children by a first marriage, all of whom are dead and concerning whom no information could be obtained. One of our patients had a daughter, who died at the age of 8 and had no pigmented lesions. A second one of our patients had 2 girls, both of whom are dead, and has 2 sons, aged 22 and 23. None of them had the pigmented anomaly.

Clinical Notes

PILOMOTOR ACTION OF NICOTINE

A New Pharmacodynamic Test of the Skin

S. ROTHMAN, M.D., AND J. M. COON, PH.D., CHICAGO

If 0.1 to 0.2 cc. of nicotine sulfate or nicotine picrate diluted with physiologic solution of sodium chloride to a concentration of 1 : 100,000 is injected intradermally into a human being, during or immediately after the injection "goose flesh" appears in an irregularly shaped area, 3 to 5 cm. in diameter, surrounding the puncture and subsides within two minutes.

Pilomotor response produced by an intradermal injection of a 1 : 100,000 solution of nicotine.

The susceptibility to the pilomotor effect of nicotine seems to parallel the tendency of "goose flesh" to develop physiologically in the subject.

The reaction fails to appear if the postganglionic sympathetic fibers of the tested area of skin have degenerated. Furthermore, it fails to appear in certain cutaneous lesions, for instance, in urticarial wheals. Local anesthetics destroy the response even in high dilutions.

The test is based on the action of nicotine on sympathetic nerve receptors in the skin. From there the impulse reaches the pilomotor endings by way of an axon reflex. Experiments on the nature of this mechanism will be published in the *Proceedings of the Society for Experimental Biology and Medicine,* the *Journal of Pharmacology and Experimental Therapeutics* and the *Journal of Investigative Dermatology.*

The same test can be performed with intracutaneous injections of acetylcholine in a concentration of 1 : 40,000 and with alpha lobeline in a concentration of

From the Section of Dermatology of the Department of Medicine and from the Department of Pharmacology, University of Chicago.

1 : 1,000,000. However, for routine tests nicotine is recommended, since it is less expensive and is stable to the heat of sterilization in the dilution used. The sterilized solution can be kept in a stoppered bottle indefinitely. As the dose of the nicotine required for the test is extremely small (1 microgram), the danger of intoxication is not to be considered. In experiments in which we were subjects, one hundred times this dose was given without the slightest symptoms.

Clinically the nicotine test was utilized first to study the restoration of pilomotor function in pedicle skin grafts.[1] The reaction can be used for testing and localizing disturbances of the sympathetic nervous system in its peripheral neurons as the histamine test (also based on an axon reflex) is used for detecting disturbances of the sensory system.

1. Kredel, F. E., and Phemister, D. B.: Recovery of Sympathetic Nerve Function in Skin Transplants, Arch. Neurol. & Psychiat. **42**:403 (Sept.) 1939.

APPARATUS TO HOLD SHOCKPROOF X-RAY CABLE

GORDON B. TAYLOR, M.D., UTICA, N. Y.

In the April 1939 issue of the ARCHIVES, Stryker[1] described a novel inexpensive apparatus to hold shockproof x-ray cables out of the way. Owing to my inability to find readily a spring of the proper tension such as he described, I adopted the following modification:

For each cable two screen door springs are used. A loop, through which the cable passes, is formed by one spring, and the ends of this loop, together with

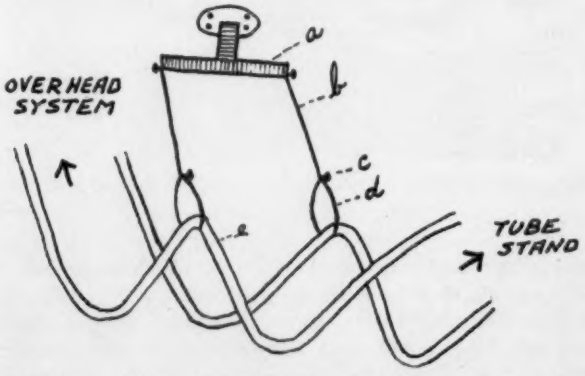


Diagram of apparatus to hold shockproof x-ray cables: *a*, cross bar suspended from ceiling; *b*, suspension spring; *c*, bolt and nut; *d*, looped spring; *e*, redundant shockproof cable.

the lower end of the second spring, are fastened by a bolt and nut. The upper end of the second spring is attached to a cross bar suspended from the ceiling. These springs provide ideal mobility and support for the cables, have the right degree of tension and are inexpensive and readily obtainable.

258 Genesee Street.

1. Stryker, G. V.: Inexpensive Apparatus to Hold Shockproof X-Ray Cables Out of the Way, Arch. Dermat. & Syph. **39**:705 (April) 1939.

ELASTIC ADHESIVE DRESSINGS IN THE TREATMENT OF LICHEN SIMPLEX CHRONICUS

GEORGE V. KULCHAR, M.D., SAN FRANCISCO

The treatment of the various circumscribed, infiltrated and lichenified areas of neurodermatitis included under the name lichen simplex chronicus is at times difficult. Cauterization with chemicals and roentgen rays, refrigeration and application of ointments containing antipruritic, keratolytic and reducing substances are the most commonly suggested forms of therapy. These are often ineffectual in eradicating the lesions or in relieving the frequently intense pruritus. Trauma inflicted on the lesions by the patient in an attempt to relieve the itching is often a considerable factor in the persistence of the lesions despite treatment. Textbooks do not mention a method of treatment which has proved effective, particularly on the extremities.

I recommend painting the involved area with a 5 per cent aqueous solution of methylrosaniline (gentian violet), which is allowed to dry. A spiral bandage of elastic adhesive tape (elastoplast) is then applied firmly, beginning well below the lesion and continuing for several inches above it, permitting each turn to overlap one-half the width of the bandage. The itching is almost always relieved at once. On removal of the dressing a week later, the epidermis at the site of the lesion is found to be softened and is easily removed with a small curet. The area is repainted and the bandage reapplied. Three or four weeks of treatment is usually sufficient, although it is well to continue with a protective dressing for several weeks after involution is complete, to avoid friction from the clothing. Bathing may be continued, and if properly applied the dressing is not particularly unsightly beneath the stocking.

This treatment has been gratifyingly effective for lesions refractory to other methods. Persistent lesions of hypertrophic lichen planus and infiltrated lichenified areas of eczema about the ankles of patients with venous varicosities may be also successfully treated by this method.

USES FOR CELLOPHANE TAPE IN OFFICE PRACTICE

JOSEPH M. SHELTON, M.D., WASHINGTON, PA.

I have found a dispenser of "Scotch" cellophane tape to be a handy office adjunct. The dispenser is of the type frequently used in department stores for sealing wrapped packages. The tape is of ½ inch (1.3 cm.) width, and any desired length can be quickly detached by the built-in cutter.

Its two principal uses in my experience have been:

1. For attaching lead foil shielding to patients' skin prior to giving roentgen treatment. The tape holds firmly for an hour or longer and comes away readily, leaving no gummy deposit on the skin.

2. For holding two pads of cotton in place and making a satisfactory eye shield for use during ultraviolet irradiation.

It is not a satisfactory substitute for ordinary adhesive tape for dressings, however, since it tends gradually to separate from moist or uneven surfaces.

From the Pittsburgh Skin and Cancer Foundation, Lester Hollander, M.D., Medical Director.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

ERYTHEMA NODOSUM WITH CONJUNCTIVAL NODULES. L. S. GREENE and M. W. PERRY, Am. J. Ophth. **22**:389 (April) 1939.

The authors report a typical case of erythema nodosum of the extensor surfaces of the extremities with nodules of the conjunctivas of both eyes. No similar cases were found recorded in the American literature, but a number of references to foreign journals are cited.

GUMMA OF THE ORBIT. MAX FINE, Am. J. Ophth. **22**:595 (June) 1939.

The author presents 2 cases of gumma of the orbit, considered chiefly from the standpoint of differential diagnosis from other conditions which may be responsible for the unilateral exophthalmos. Tabular data on 24 cases reported in the literature are recorded.

WIEDER, Milwaukee.

THE MINIMAL EFFECTIVE CONCENTRATIONS OF ARSENIC AND BISMUTH COMPOUNDS ON T. PALLIDUM IN VITRO IN RELATION TO THE THERAPEUTIC DOSE. H. EAGLE, Am. J. Syph., Gonor. & Ven. Dis. **23**:310 (May) 1939.

The antispirochetal action of arsenic and bismuth compounds in vitro was determined in the presence of the edema fluid from a chancre of a rabbit, which presumably corresponds in composition to the intercellular fluids at the inflammatory syphilitic focus. Under these conditions, "arsenoxide" (metaaminoparahydroxyphenylarsenoxide) had a definite antispirochetal action within one to two hours at room temperature in concentrations of approximately 1 : 1,000,000 to 1 : 400,000,000; arsphenamine, neoarsphenamine and silver arsphenamine were similarly effective in concentrations of 1 : 250,000 to 1 : 1,250,000, and two water-soluble bismuth compounds were effective in concentrations of 1 : 50,000 to 1 : 225,000. As the period of observation is prolonged, the concentration of the various compounds necessary to immobilize the organisms decreases decidedly. It follows, therefore, that the minimal effective concentrations just cited are several times greater than those which would have been necessary had the experiments been carried out for eight hours at 37 C.

The author was able to correlate satisfactorily the experimentally determined minimal effective concentrations of arsenic and bismuth compounds in vitro and those which may be attained in the tissue fluids after their therapeutic administration.

SYPHILIMETRY WITH THE EXCLUSION SLIDE TESTS IN 44 CASES OF EARLY TREATED SYPHILIS. B. LEVINE, S. LITTMAN and B. S. KLINE, Am. J. Syph., Gonor. & Ven. Dis. **23**:413 (July) 1939.

The authors emphasize that it is the sensitive blood tests and not demonstration of Spirochaeta pallida which, at present, are of the greatest value in the early detection of syphilis.

The microscopic slide precipitation tests for the exclusion of syphilis, reacting with a very small amount or more of reagin, have been found to serve well as criteria of the presence or absence of the disease. The exclusion slide tests were also found to be more satisfactory in the control of adequate syphilitic treatment than was therapy by schedule. In 44 cases of early treated syphilis the blood tests were frequently repeated, and when at least three consecutive exclusion slide

tests at intervals of a week or more gave negative results an examination of the spinal fluid was made, and if the result of this was negative for evidence of syphilis treatment was discontinued. The average period of treatment (continuous, alternating arsenicals and bismuth compounds) was twenty-three months, the minimum five weeks and the maximum over five years. The patients have been followed for an average of four years and ten months and have been found to date to be free of all signs and symptoms of the disease.

LOWERED RESISTANCE TO SYPHILITIC INFECTION IN OVARIECTOMIZED RABBITS. C. K. HU, Am. J. Syph., Gonor. & Ven. Dis. **23**:446 (July) 1939.

It was found that removal of the ovaries caused syphilitic infection in rabbits to run a more severe course than in animals with the ovaries intact. The estrogen hormone exerts an inhibitory influence on the course of the infection, but it alone is not responsible for all the difference in severity of the infection between the two sexes.

ERYTHEMA OF THE NINTH DAY FOLLOWING ARSPHENAMINE THERAPY. NORMAN N. EPSTEIN and EDWARD A. LEVIN, Am. J. Syph., Gonor. & Ven. Dis. **23**:490 (July) 1939.

Six cases of nine day erythema are reported. Five of the patients were syphilitic and 1 was nonsyphilitic. The clinical features of this reaction are an erythematous generalized morbilliform eruption with congestion of the mucous membranes and general adenopathy. Constitutional symptoms are variable. Fever is constant at the onset. Leukocytosis may be present. In the series studied the average duration of the reaction was five days. In all instances neoarsphenamine was the drug employed. Uncomplicated erythema of the ninth day is not a contraindication for further arsenical therapy. The initial dose after the reaction should be considerably reduced.

REUTER, Milwaukee.

DERMATOMYOSITIS WITH SCLERODERMA, CALCINOSIS AND RENAL ENDARTERITIS ASSOCIATED WITH FOCAL CORTICAL NECROSIS. J. H. TALBOTT, E. A. GALL, W. V. CONSOLAZIO and F. S. COOMBS, Arch. Int. Med. **63**:476 (March) 1939.

The authors report an interesting case in which a presumptive diagnosis of Addison's disease was based on a history of profound weakness, malaise, gastrointestinal disturbances, loss of weight and pigmentation of the skin. During the treatment for this condition the skin became shiny, taut and atrophic, and the presence of dermatomyositis with scleroderma became apparent. Eleven months after the onset of symptoms and three months before death subcutaneous nodules were palpable, and roentgen examination revealed calcinosis. At postmortem examination the adrenal glands were normal in size and appearance. The significant anatomic diagnoses were chronic nonsuppurative dermatomyositis, scleroderma, calcinosis and obliterative endarteritis of the kidneys.

As it is frequently difficult clinically to differentiate scleroderma and dermatomyositis, and in consideration of the observations at autopsy, the authors expressed the opinion that such a differentiation was futile in this instance. The similarity of the histologic changes in these two conditions suggested that there was a common cause.

A VARIABLE SYMPTOM COMPLEX OF UNDETERMINED ETIOLOGY WITH FATAL TERMINATION. E. C. REIFENSTEIN, E. C. REIFENSTEIN JR. and G. H. REIFENSTEIN, Arch. Int. Med. **63**:553 (March) 1939.

The authors select from the literature reports on 17 patients each with a symptom complex having all or many of the following features: prolonged fever, polyarthritis, polyserositis, endocarditis, erythematous cutaneous lesions, nephritis,

anemia and a remittent cachectic course. They report an additional case in which there were most of these features and several additional pathologic changes.

The condition under discussion is regarded by the authors as a symptom complex having no definite diagnostic criteria, no known etiologic factor and no single name by which it is designated. It has been described under the names of "visceral manifestations of the erythema group" (Osler), disseminated lupus erythematosus, "atypical verrucous endocarditis" (Libman and Sacks), fever of unknown origin (Christian) and diffuse peripheral vascular disease (Baehr and others). In order to further the conception of this symptom complex as a unity, it is suggested that similar cases in the future be considered as a group, so that their various peculiarities may be given equal consideration but not undue prominence.

COMPARATIVE EFFECTS ON EARLY SYPHILIS OF COMBINED AND OF ALTERNATING TREATMENT. WALTER BECKH and CHARLES W. BARNETT, Arch. Int. Med. **63**:974 (May) 1939.

The authors report the results in patients with early syphilis given the combined treatment with iodobismitol (seventy injections in seventy-five weeks) and neoarsphenamine (forty injections in the first forty-five weeks of treatment). These results are compared with those obtained by the alternating method (thirty injections of neoarsphenamine in three courses of ten each and sixty injections of iodobismitol in three courses of twenty each, and the two drugs simultaneously for the first ten weeks).

The frequency of serologic reversal at the end of five and of eighteen months of treatment was slightly greater for the patients given combined treatment than for those given the alternating type. The incidence of abnormal cerebrospinal fluids was the same in the two series. The incidence of relapse, both clinical and serologic, was greater among the patients given combined treatment than among those given the alternating type.

Beckh and Barnett believe that any further use of combined therapy is undesirable since they regard prevention of relapse as the most important indication of efficiency of antisyphilitic therapy.

A SYNDROME CHARACTERIZED BY PREMENSTRUAL DERMATOSIS, POLYHYPERMENORRHEA, DYSMENORRHEA, MENSTRUAL HEADACHE AND AN INCREASED URINARY EXCRETION OF SODIUM PREGNANEDIOL GLUCURONIDE. E. C. HAMBLEN, Endocrinology **24**:269 (Feb.) 1939.

Hamblen describes a patient whose symptoms included cyclic ecchymotic lesions during the latter part of the progestational phase of the menstrual cycle, polyhypermenorrhea, dysmenorrhea and menstrual headache. The dermatosis recurred each month about ten days before an expected period and practically disappeared when bleeding began. The lesions were scattered over the upper portions of the arms and thighs and were painful and itchy for the first day.

Intensive estrogenic therapy resulted in the disappearance of all the symptoms.

HAIR GROWTH FOLLOWING THE REMOVAL OF A HYPERNEPHROMA. EMERY SCHILL, Endocrinology **24**:572 (April) 1939.

A patient 68 years of age had lost all his hair several years before an operation for removal of a hypernephroma. Two weeks after the operation new growth of hair began to appear sparsely on the head and face and gradually increased. This growth of hair persisted; five years later a recurrent tumor of the vertebral column did not influence the growth. Schill suggests that adrenocortical tumors may exert a feminizing action (in addition to the usual masculinizing influence) or that in this case the pressure of the tumor on the adrenal gland inhibited its endocrine function.

LYNCH, St. Paul.

DIPHTHERITIC INVOLVEMENT OF THE LIPS, WITH ABSENCE OF SIGNS IN THE NOSE AND THROAT. H. J. LAVENDER and J. B. SQUIRES, J. A. M. A. **111**: 915 (Sept. 3) 1938.

Lavender and Squires report the case of a Negress aged 10 years in whom, during treatment for an extensive burn from fire, an infection of the lower lip developed. There was swelling, and the surface was covered by a whitish membrane. Similar lesions developed on the right buccal mucous membrane and later on the upper lip. Ulceration and sloughing of the lesions on the lips occurred. Klebs-Loeffler bacilli were demonstrated in cultures of material from the lips, from the buccal mucous membrane and from the sockets of teeth which had loosened and had been extracted by the patient. The nose and throat showed no evidence of diphtheritic involvement, and repeated cultures for Klebs-Loeffler bacilli gave negative results until after the twenty-third day. There was a positive reaction to a virulence test with Klebs-Loeffler bacilli from the lips and buccal mucous membrane. The patient recovered with complete healing of the ulcerations.

COCCIDIOIDOMYCOSIS: THE PRELIMINARY ACUTE INFECTION WITH FUNGUS COCCIDIOIDES. E. C. DICKSON, J. A. M. A. **111**:1362 (Oct. 8) 1938.

According to Dickson, primary infection with Coccidioides immitis is usually acquired by inhalation. The primary acute infection of the respiratory tract is known in the San Joaquin Valley as valley fever or desert fever when it is accompanied by erythema nodosum; most patients recover without complications. In a small percentage of cases the disease may be chronic; in such cases granulomatous lesions develop in various tissues of the body (granuloma coccidioides). At the onset, headache is usual. General aches and pains appear, sometimes referred to the chest. Sore throat, conjunctivitis and gastrointestinal disturbances are common. Chills and sweating may occur; the fever is usually not high. The bronchitic symptoms are variable. The lesions of erythema nodosum usually appear in from eight to fifteen days after the onset and disappear after four or five days. Roentgenograms of the chest during the acute attack show dense shadows in the hilar regions and other parts, indicating both enlargement of the hilar glands and parenchymatous involvement. These shadows usually promptly disappear with recovery. Dickson found Coccidioides immitis in 72 of 112 specimens of sputum sent to him from patients suffering from valley fever. Eosinophilia may occur. Dickson found the coccidioidin cutaneous test to be helpful and of diagnostic import; patients with valley fever were much more reactive to the test than patients with the later forms of the infection.

LEPROSY OF THE UPPER RESPIRATORY TRACT: DISCUSSION OF EARLY AND MODERATELY ADVANCED CASES. F. J. PINKERTON, J. A. M. A. **111**:1437 (Oct. 15) 1938.

Pinkerton reviews his experience in Hawaii in the study of leprous patients for lesions in the upper respiratory tract. He states that practically every patient with leprosy has some nasal lesion due to the disease. Dryness, stuffiness, excessive crusting, blocking and epistaxis are common symptoms. The nasal bone is never destroyed, but the nasal cartilage commonly shows the typical depression at the tip of the nose; in a patient with syphilitic saddle nose the depression is due to destruction of the bony septum. The pharynx is seldom attacked. Nodules, leukoplakia and diffuse thickening may be lingual manifestations of leprosy. The tonsils were found to be affected in 20 per cent of cases, and in 40 per cent of cases of moderately advanced nodular involvement there were leprous lesions of some kind in the larynx. Huskiness may be an early symptom. In making a nasal "snipping" for bacteriologic examination, scraping hard enough to get some of the epithelium is essential, but one should not scrape hard enough to draw blood, as this interferes with the further examination. The scrapings are stained by the Ziehl-Neelsen technic. The favorite site for scraping is the septum, near

the anterior border, or on the side of the nose immediately above the anterior end of the inferior turbinate. According to Pinkerton, more than 40 per cent of leprous patients with nodular lesions show a positive Wassermann reaction. With progression of the disease, the tendency to positivity of the reaction increases. In the neural types the Wassermann test is not specifically affected. Pinkerton has not observed any specific therapeutic effect from chaulmoogra oil U. S. P. Leprous lesions in the upper respiratory tract are best treated by measures to improve the general health, the use of bland oil sprays and hygienic care.

OCCUPATIONAL DERMATOSES: AN EDUCATIONAL PROGRAM. C. GUY LANE, J. A. M. A. **111**:1521 (Oct. 22) 1938.

Since diseases of the skin due to occupation are assuming increased importance, Lane stresses the need for cooperation between interested authorities to provide adequate training in the subject for physicians and nurses. In this way prophylaxis may be possible in many cases, and improvement in the care of the patients will ensue.

INCIDENCE OF OCCUPATIONAL DERMATOSES AND THEIR CAUSES IN THE BASIC INDUSTRIES. L. SCHWARTZ, J. A. M. A. **111**:1523 (Oct. 22) 1938.

According to the reported statistics of nine states, the incidence of occupational dermatoses in the United States is about 65 per cent of cases of occupational disease. Schwartz states that in a study made by the Public Health Service among more than 100,000 workers in various factories, it was found that about 1 per cent of all the workers were affected with occupational dermatoses in one year. Thirty per cent of all cases are observed in the metal industries, the domestic and food industries being second and third respectively. The leading chemical causes in the decreasing order of frequency are as follows: Alkalis, petroleum products, solvents, plants, metals and metal plating, acids (including chromic acid), dyes, rubber compounds and resins.

INDUSTRIAL DERMATITIS: DEFINITIONS AND CRITERIA FOR DIAGNOSIS. M. B. SULZBERGER and C. W. FINNERUD, J. A. M. A. **111**:1528 (Oct. 22) 1938.

Industrial dermatitis is defined by Sulzberger and Finnerud as "an inflammatory condition of the skin incidental to systematic work or labor or habitual employment, especially in the productive arts or manufactures." They list the criteria of primary or secondary importance in its diagnosis. The physician must confine his testimony in court to the purely medical phase and must not attempt to make legal interpretations in a case under dispute. Sulzberger and Finnerud point out that the appearance and course of dermatitis, industrial or not, are often not characteristic, indicative or diagnostic of the actual causal agent. Polyvalent sensitizations or synergistic reactions may prove confusing and impossible of logical solution.

PRACTICAL ASPECT OF THE PREVENTION OF INDUSTRIAL DERMATOSES. E. D OSBORNE and J. W. JORDON, J. A. M. A. **111**:1533 (Oct. 22) 1938.

Industrial dermatitis may be due to a primary irritant or to sensitization. Many substances used in industry are capable of producing dermatitis by both methods. When the condition is due to a primary irritant some mechanical means of protection and education of the worker will usually result in prevention of the dermatitis. With dermatitis due to sensitization, prophylaxis is difficult; at present it is impossible in most cases. Osborne and Jordon do not favor preemployment performance of patch tests, nor do they describe any type of skin as ideal for general employment in industry. Certain compounds which have a high sensitizing index may be eliminated in favor of less harmful compounds. Prevention of exposure to known sensitizing substances may be attained by improved

sanitary devices, proper clothing, provision of proper cleansing facilities and instruction to workers regarding the danger in their occupation. Early recognition of sensitization dermatitis is considered of extreme importance.

ANALYSIS OF CLAIMS IN INDUSTRIAL DERMATOSES. J. G. DOWNING, J. A. M. A. **111**:1536 (Oct. 22) 1938.

Of a total of 2,000 claims for compensation examined by Downing for insurers, the dermatosis was considered to be nonoccupational in 845 cases and occupational in 1,004. In 151 the origin of the rash was not established. In many instances interesting and puzzling situations developed, and Downing cites cases to illustrate medical procedure and the interpretations on evidence as decided by the lay board appointed under the Massachusetts Workmen's Compensation Act.

COMPENSATION LAWS AND RELATED MEDICOLEGAL CONSIDERATIONS. H. R. FOERSTER, J. A. M. A. **111**:1542 (Oct. 22) 1938.

Foerster points out that if compensation laws are so liberally interpreted that the consideration of liability is not determined, a condition of socialized medicine exists. On the other hand, the physician must fulfil his obligations so that a workman injured in the course of his employment will receive compensation. The dermatologist should be familiar with the compensation laws and their interpretation and application. There has been a steady development toward the liberal, or progressive, side. Variation exists in the laws of the different states. Foerster favors the laws in states where compensation is provided for patients with nondisabling diseases as well as for patients who are totally disabled, since it permits early examination and more effective therapy. If a dermatologist is not consulted before disability has occurred, the correct diagnosis may be difficult. Foerster is of the opinion that physicians should be assigned more than in the past to interpret the medicolegal aspects of compensation cases.

TREATMENT OF CANCER OF THE EYELIDS. G. S. SHARP, J. A. M. A. **111**:1617 (Oct. 29) 1938.

Sharp reports uniformly successful treatment by means of irradiation in 62 consecutive cases of cancer of the eyelids. Biopsy was performed in 48 instances. Sharp advocates a histologic diagnosis to differentiate a benign from a malignant process. The radium was screened to exclude the beta rays and was applied on the surface of the lesion, interstitially or in a combined manner, all according to the type and size of the lesion.

BENIGN GRANULOMA OF THE NOSE (SO-CALLED SARCOID). F. B. KISTNER and T. D. ROBERTSON, J. A. M. A. **111**:2003 (Nov. 26) 1938.

The case of a woman aged 50 is reported in which deep tissue from the bone at the junction of the nasal bones and the ascending process of the superficial portion of the maxilla and from the nasal mucosa was taken for biopsy and revealed structure typical of sarcoid. Later a purplish, plaquelike infiltrative process involved the skin of practically the entire nose. This also was considered sarcoid.

LEWIS, New York.

THE MODE OF ACTION OF SULFANILAMIDE ON STREPTOCOCCUS. F. P. GAY, A. R. CLARK, J. A. STREET and D. W. MILES, J. Exper. Med. **69**:607 (May) 1939.

The result obtained on adding sulfanilamide to the streptococcus in the test tube is usually bacteriostasis and not complete destruction of even small numbers of bacteria.

As a result of experimental studies, the authors conclude that sulfanilamide not only does not completely destroy the streptococcus but does not even impair

its innate virulence. It acts on the streptococcus by inhibiting growth and by a temporary inhibition of hemotoxin formation, but only under certain conditions. Sulfanilamide differs from many other drugs in that it is not fixed or absorbed by the streptococcus.

LYNCH, St. Paul.

INVESTIGATIONS ON THE POSSIBLE CARCINOGENIC EFFECT OF ANTHRACENE AND CHRYSENE AND SOME OF THEIR COMPOUNDS. J. A. POLLIA, J. Indust. Hyg. & Toxicol. **21**:219 (June) 1939.

When painted on the skin of mice, the author found that 1 : 2 : 5 : 6-dibenzanthrene produced warts which in all animals surviving one hundred and thirty-two to one hundred and fifty-five days were found to be malignant; chrysene, anthracene, oil-soluble chrysene red and water-soluble chrysene blue and green failed to produce any changes when used in the same manner.

WIEDER, Milwaukee.

A CASE OF AGRANULOCYTOSIS OBSERVED DURING THE TREATMENT OF SYPHILIS. J. BRANTS, Dermat. Wchnschr. **106**:696 (June 18) 1938.

Brants reports a case of relatively mild agranulocytosis apparently due to the syphilis rather than to the treatment with bismuth or neoarsphenamine.

NONSPECIFIC WASSERMANN REACTION OF BLOOD AND SPINAL FLUID DUE TO CONTAMINATION WITH BACILLUS SUBTILIS. A. POHLNER, Dermat. Wchnschr. **106**:701 (June 18) 1938.

Contamination of whole blood with bacillus subtilis before removal of the serum may cause a nonspecific positive Wassermann reaction. In 27 of 28 non-syphilitic patients positive Wassermann reactions were obtained in spinal fluids contaminated with the same organism.

LIVER DAMAGE RESULTING FROM TREATMENT WITH SULFANILAMIDE OF CHRONIC RECURRING ERYSIPELAS. WOLFGANG GERTLER, Dermat. Wchnschr. **106**:725 (June 25) 1938.

Subacute yellow atrophy of the liver developed in a patient who had taken 140 tablets of sulfanilamide of 0.3 Gm. each. The drug was prescribed in doses of 2 to 4 tablets daily, as a prophylactic for recurring erysipelas. It is concluded that this drug should not be prescribed over long periods without periods of rest and close supervision.

POSSIBILITY OF TESTS FOR SYPHILIS ON DRIED BLOOD AND SERUM. T. LINK, Dermat. Wchnschr. **106**:729 (June 25) 1938.

The results of 730 Wassermann tests for syphilis made on dried blood by the Chediak method closely followed those obtained by standard methods on the corresponding serums.

TAUSSIG, San Francisco.

THE CLINICAL IMPORTANCE OF THE SEROLOGIC TUBERCULOSIS TESTS FOR CUTANEOUS DISEASES AND EPIDIDYMITIS. R. BRANDT and G. KIEN, Dermat. Ztschr. **77**:57 (Feb.) 1938.

The authors used the Witebsky-Klingenstein-Kuhn antigen in a large number of complement-binding and agglutination tests. The material included serums from 321 patients with various dermatoses but with no clinical signs of cutaneous or organic tuberculosis. Fourteen (4.3 per cent) of these serums gave positive reactions. Of a total of 151 patients with cutaneous tuberculosis, 72 gave positive reactions, among whom were only 2 of 16 with glandular tuberculosis and scrofuloderma. Of 94 patients with lupus erythematosus, 29 (30.9 per cent) gave positive reactions, while in 46 (13.5 per cent) of 146 with acne vulgaris, the reactions were

positive. In all but 1 patient with epididymitis which was proved to be tuberculous, the serologic reactions were positive. A negative reaction could thus be considered strong evidence against the tuberculous causation of epididymitis.

RESTORATION OF FUNCTION OF THE SWEAT GLANDS IN CUTANEOUS TRANSPLANTS. LUDWIG GUTTMANN, Dermat. Ztschr. **77**:73 (Feb.) 1938.

The patient had had an injury of the palm repaired by a skin graft taken from the trunk. He noted that perspiration first occurred on the graft at the time that sensitiveness was restored. By means of the colorimetric method of Minor, it was noted that the quality of the perspiration of the graft was similar to that of the area from which it was obtained rather than to that of the other palm.

TREATMENT OF LUPUS VULGARIS WITH GRENZ RAYS. FRIEDRICH KALZ, Dermat. Ztschr. **77**:232 (April) 1938.

The author agrees with a number of European writers that the grenz rays are particularly suitable for the treatment of lupus vulgaris. He has used doses not exceeding 3,000 roentgens, with 7 cm. target skin distance, 12.5 kilovolts and 15 milliamperes, with decided success. Slight atrophy may develop, but this is considered justifiable. The one objection is that when the disease recurs further treatment by any method is difficult on account of the cutaneous damage.

RECENT VIEWS ON THE ORIGIN OF NEVI MOLLES AND THE SOFT NEVUS CELLS. ERICH HOFFMANN, Dermat. Ztschr. **77**:297 (May) 1938.

Hoffmann discusses the various theories concerning the source of the nevus cells. He concludes, with the majority of modern authors, that these cells are derived from the Schwann cells.

INJURY OF THE HAIR SHAFT. ŠIME ČAJKOVAC, Dermat. Ztschr. **77**:305 (May) 1938.

A woman who had had her hair bleached, shampooed and waved frequently complained of loss of hair. Examination showed that the hair was broken off. Microscopically there was profound alteration in the structure of the hair, particularly a loss of medulla with annular segmentation. The author made extensive investigations on the effect of various cosmetic procedures on hair and was able to show that a decided loss of elasticity and strength resulted from these procedures.

TAUSSIG, San Francisco.

RADIUM TREATMENT OF CUTANEOUS CAVERNOUS HEMANGIOMAS (SURFACE APPLICATION OF RADIUM TUBES IN GLASS TUBES). MAGNUS STRANDQUIST, Acta radiol. **20**:185, 1939.

Three hundred cases of cavernous hemangioma were treated at Radiumhemmet during the years 1936 and 1937 by gamma radiation according to the technic described by the author (*Acta radiol.* **1**:1 to 15, 1939). No injuries from treatment were noted. Fifteen of the 300 angiomas were ulcerated before treatment; all of these disappeared entirely but left scars the size of the areas of ulceration. In 72 per cent of the other 285 cases there was complete disappearance of the lesions without scar; in 14 per cent there was improvement without changes of the skin, and in 14 per cent there was complete disappearance of the lesions with pigmentary irregularity or slight telangiectasia. The article describes the author's method in detail. It is based on calculation of the average dose for 1 cm. of superficial tissue layer. This is considered definitely superior to the method of estimation of dosage by surface effect. WIEDER, Milwaukee.

Society Transactions

LOS ANGELES DERMATOLOGICAL SOCIETY

KENDAL FROST, M.D., *Chairman*

March 14, 1939

CHARLES R. CASKEY, M.D., *Secretary*

A Case for Diagnosis. Presented by DR. PAUL D. FOSTER.

R. F., a white man aged 66, bumped his head against a door seventeen years ago and incurred a swelling, which, however, subsided. Since that time a soft mass has been present, which has shown decided growth during the past three years. At the present time there is an egg-sized soft swelling on the left side of the head. No pain is complained of, but the lesion is erythematous at times. The results of a serologic test were negative.

DISCUSSION

DR. CHARLES R. CASKEY: I believe a diagnosis of gumma should be considered.

DR. J. R. SCHOLTZ: The firm border which is felt in this lesion is similar to that felt in subperiosteal hematomas. I suggest considering the possibility of a tumor arising from the periosteum.

DR. PAUL D. FOSTER: The lesion was a soft elevated tumorous mass obviously under the skin. It was thought to be a lipoma on first examination. The first biopsy specimen was not diagnostic, and so a deeper section was taken, down to the periosteum of the frontal bone; but this also was not diagnostic. Although the lesion has been present for seventeen years, in my opinion the rapid growth for the past three years would justify complete removal.

A Case for Diagnosis. Presented by DR. THOMAS W. NISBET.

E. C., a Chinese girl aged 9 years, presents a purpuric eruption on the inner surface of the thighs and to a lesser extent about the waist. The eruption began about five months ago. At that time she was taking a preparation of papaw and bile salts as a laxative and continued to do so, but the eruption has gradually extended downward to the knees, and the superficial veins have become increasingly prominent. She gives a history of a rapid gain in weight. The results of Wassermann and Kahn tests were negative.

DISCUSSION

DR. NELSON PAUL ANDERSON: I suggest that the possibility of leprosy be investigated. There are peculiar lesions on the outer aspects of the eyebrows. The girl has been in this country only six months, and the eruption has been present about the same length of time. The eruption is in a location in which I did not care to test the sensitivity of the skin.

DR. PAUL D. FOSTER: The girl has been in this country only for the past five months. She has gained 21 pounds during that time. At present she is fat, whereas I understand that previously she appeared emaciated. I believe this to be a case of striae cutis distensae resulting from rapid increase in weight.

DR. H. C. L. LINDSAY: I agree with Doctor Foster.

DR. SAMUEL AYRES JR.: I am inclined to believe that the case is one of striae atrophicae. I think the rapid gain in weight which causes the separation of the elastic fibers finally produces atrophic striae, which later become white and scarlike.

DR. J. R. SCHOLTZ: The lesions resemble those of striae cutis distensae. With the patient's remarkable gain in weight at her age, the diagnosis of pituitary basophilic adenoma should also be considered. Of course, other observations would be necessary to establish this diagnosis.

Lymphogranuloma Venereum. Presented by DR. S. O. CHAMBERS and DR. A. J. GRECO (by invitation).

B. L. J., a Negro aged 40, says that he had a gonococcic infection in 1918. In 1921 there was an inguinal bubo on the left side but no sore. In November 1938 he noticed a small sore on the frenum, which appeared a day after illicit intercourse. The ulcer gradually enlarged and was painted with iodine, which made it worse. He then noticed an ulcer near the meatus and one on the prepuce. He went to the Los Angeles General Hospital the last week in November 1938. At that time the penis showed an ulcer on the dorsum of the prepuce which penetrated between the outer and the inner surface. There was a smaller ulcer on the frenum, and a small puckered scar was seen on the glans below the meatus. In February 1939 several dark field examinations for Spirochaeta pallida gave negative results; also smears were negative for Ducrey's bacillus. Serologic tests repeatedly gave negative results. The result of a Frei test was positive on February 16. With the area under procaine hydrochloride anesthesia, a portion of the ulcer was removed for biopsy. The infection spread, involving the sites of suture, and subsequently the ulcer increased in size.

Treatment was given as follows:

Feb. 15, 1939. Soaks in 1:4,000 solution of potassium permanganate.

Feb. 16. Sulfanilamide, 1.3 Gm. three times a day for three days.

Feb. 23. Soaks in solution of potassium permanganate.

Feb. 27. Sulfanilamide, 1.3 Gm. three times a day for three days and soaks in solutions of potassium permanganate.

March 2. Sulfanilamide, 1.3 Gm. twice a day for one week and soaks in solution of potassium permanganate.

The ulcer on the frenum healed, but the one on the scar left by removal of tissue for biopsy is slowly enlarging.

DISCUSSIONN

DR. THOMAS W. NISBET: I do not think these lesions are typical of venereal lymphogranuloma. The characteristic suppuration of the deep inguinal glands is not present, and the primary lesion is entirely too large. In most cases of venereal lymphogranuloma the initial lesion is so small as to pass unnoticed. I suppose, however, that if the Frei reaction is positive I must agree with the diagnosis.

DR. IRVING BANCROFT: For the last several years physicians have been trying to find the initial lesion of lymphogranuloma and have never been able to do so. A great many chancroids which reacted just like this have been seen. I think it is difficult to make a diagnosis without glandular enlargement.

Pigmented Nevus, with Signs of Hyperplasia. Presented by DR. H. C. L. LINDSAY.

H. M. P., a white man of 35, ten years ago noticed a brown pigmented spot in the left eyebrow, about the size of a pinhead. Five years ago the patient noticed a slight increase in size and more recently a further enlargement, until now it has a diameter of 1 cm.

DISCUSSION

DR. SAMUEL AYRES JR.: I do not think the lesion is malignant. It is a light brown flat naevus pigmentosus. He has had it for ten years. In view of the complete lack of elevation and its light color, I should freeze it with solid carbon dioxide. I have treated similar lesions with good results.

DR. CHARLES R. CASKEY: I agree with that opinion. I should not consider removal, surgically or by cautery, of a lesion that looks as innocent as that, because of the disfigurement.

DR. J. R. SCHOLTZ: It seems to me that the lesion would be better classed as lentigo, since there is simple pigmentation without any change in the consistency or contour of the skin. One might venture the guess that no nevus cells would be found in a histologic section.

DR. L. F. X. WILHELM: I should reassure the patient that the lesion is not malignant and watch it carefully over a period of a year or more.

DR. H. C. L. LINDSAY: I have reviewed some of the literature concerning melanoma, melanocarcinoma and nevocarcinoma. "A great deal of confusion exists relative to the exact nature of malignant neoplasms that arise from pigment forming tissues. Formerly most of these growths were thought to be sarcomas; now most of them are considered carcinomas" (MacKee, G. M., and Cipollaro, A. C.: Cutaneous Cancer and Precancer: a Practical Monograph, New York, The American Journal of Cancer, 1937).

Is the lesion in this case a sarcoma or a nonmalignant pigmented nevus? There is no induration and but slight color. There is not a physician here who has not at some time seen a somewhat similar lesion of long standing and seemingly nonmalignant aspect suddenly present symptoms of malignancy. Should this growth be removed? Some experts contend that such lesions should be surgically removed deeply, widely and as early as possible. Radium and roentgen ray radiation in lethal doses are advocated by others, especially before excision. Coste (cited by MacKee and Cipollaro) stated that he had obtained cures in 99 per cent of his patients with ordinary cutaneous carcinomas and in 80 per cent of those with melanomas. He administered to the tumor from 3,500 to 4,000 r of unfiltered roentgen rays at 110 kilovolts at one sitting and repeated the dose for recurrences.

DR. PAUL D. FOSTER: Dr. Samuel Becker recently saw a patient at the White Memorial Hospital with a condition which was in many respects similar to this. An area of skin approximately ¼ to ⅜ inch (0.6 to 0.9 cm.) in diameter would become black and indurated and remain so for from three to six months before another area immediately adjacent to the original one would become pigmented and go through the same procedure of becoming black and indurated. A biopsy specimen was taken from the edge of that lesion, the area which showed only pigmentation, and it was decided that the tissue was definitely malignant.

Bilateral Herpes Zoster. Presented by DR. NELSON PAUL ANDERSON.

J. C., a white woman at the age of 37, gives a history of exposure to chickenpox one month ago. About two weeks ago she noticed a lesion on the left side of the trunk, and a week later, a group of vesicles on the right side of the trunk. At present there are on the left side of the trunk a group of typical herpes zoster vesicles in a single ovoid patch and fifteen to twenty crusted lesions and on the right side, two small groups of vesicles following a nerve trunk.

DISCUSSION

DR. H. C. L. LINDSAY: I understand that herpes zoster is more frequent during an epidemic of chickenpox. I wonder if any one has noticed any increase in the incidence of chickenpox lately.

DR. SAMUEL AYRES JR.: I also had occasion to look up the literature on herpes zoster. It has been pointed out by Wise and Sulzberger (Year Book of

Dermatology and Syphilology, Chicago, Year Book Publishers, Inc., 1937, p. 212) that herpes zoster is probably a modified form of chickenpox. The fluid from a vesicle inoculated into a child will produce chickenpox, and inoculated into an adult, herpes zoster. The adult has usually had chickenpox in childhood and therefore has established a certain amount of immunity to it, and the picture of herpes zoster represents an allergic type of reaction to the chickenpox virus.

DR. IRVING BANCROFT: I have seen 15 patients with herpes zoster recently among persons being treated for syphilis. I wonder if any one else has noticed more frequent herpes in those who are being treated for syphilis.

DR. NELSON PAUL ANDERSON: I brought the patient to this meeting to prove that when herpes zoster spreads around the trunk the patient does not necessarily die.

Lichen Spinulosus. Presented by DR. SAMUEL AYRES JR. and DR. JOHN ROGERS (by invitation).

D. C. O., a boy aged 14, had an eruption on his skin which began about eighteen months ago with the appearance of follicular papules on the trunk and limbs. At present he shows acuminate follicular keratotic lesions on the trunk. There are no subjective symptoms. The blood count and the result of urinalysis were normal. The Wassermann and Mantoux reactions were negative.

DISCUSSION

DR. SAMUEL AYRES JR.: I think that this is a case of typical lichen spinulosus, which is not very common. The condition probably represents some toxic disturbance. There is frequently confusion between lichen spinulosus and lichen scrofulosus. The definite spiny lesions in this particular case serve to differentiate it. The lesions in lichen scrofulosus are redder and rounded.

DR. J. R. SCHOLTZ: As almost all the lesions which have been attributed to vitamin A deficiency are of this type, I suggest that the nutritional history be studied. My associates and I have seen this condition a number of times in persons of the social-economic stratum to which the patient belongs.

Squamous Cell Carcinoma of the Tongue, Treated as Gumma. Presented by DR. ARTHUR FLETCHER HALL.

E. W. G., a white man aged 47, in August 1938 noticed an infiltrated fissure in the midportion of the right side of his tongue. This grew so rapidly that his physician had a serologic test made, the result of which was strongly positive. Antisyphilitic treatment was instituted and continued until February 1939. When seen in March, the lesion on the right side of the tongue showed a smooth irregular infiltration of the anterior half of the right side of the lingual margin; the posterior half showed a lumpy and cauliflower-like firm mass with a definite border. The lymph glands of the right side of the neck were palpable and tender. The diagnosis from a histologic section was squamous cell carcinoma.

I assumed that gumma did exist. The whole anterior half of the lesion resolved considerably with antisyphilitic treatment. This is an example of what omitting a histologic examination can do.

DISCUSSION

DR. NELSON PAUL ANDERSON: A considerable percentage of malignant growths of the tongue are associated with tertiary syphilis of that organ.

DR. L. F. X. WILHELM: I should like to report an interesting case I observed in 1925 while associated with Dr. Fordyce in New York. Tuberculosis developed in a man at the age of 19 or 20, for which he went South. Apparently that condition was arrested, but he contracted syphilis, for which he went to New York for treatment. While he was under treatment, a nodular lesion appeared on the

tongue. It was definitely malignant, as proved by biopsy. One third of the tongue was involved, and so it was excised. I have seen this man in Los Angeles recently, and apparently he has been cured of tuberculosis, syphilis and cancer.

DR. SAMUEL AYRES JR.: I suggest radium or contact roentgen ray treatment for such lesions.

KENDAL FROST, M.D., *Chairman*
April 11, 1939
CHARLES R. CASKEY, M.D., *Secretary*

Blastomycosis (Probable). Presented by DR. NELSON PAUL ANDERSON and DR. O. MILTON STOUT (by invitation).

E. Q., a Mexican laborer aged 35, states that his first lesion appeared in the right groin when he was 5 years old. It began as a small papule and rapidly grew to the size of a dollar. Three months after the appearance of the first lesion similar lesions appeared and in the course of time healed, leaving scars. New lesions always appeared in the borders of scars. The lesion now present in the upper posteromedial aspect of the right thigh appeared in 1934. At present the patient presents an elevated papillomatous fungating lesion about the size of a silver dollar in the right groin. Extending from the midline to the posterior axillary line of the abdomen is a narrow elevated patch covered with adherent crusts. The borders are purplish red, shading gradually into the color of normal skin. A few small miliary abscesses are seen. On the upper medial aspect of the right thigh are two granulomatous crusted areas. In 1922 antisyphilitic treatment was instituted, and although the serologic reactions were negative and there was no history of primary or secondary syphilitic infection, the patient has had irregular treatment to 1935.

DISCUSSION

DR. MAX WOLFF: I think Dr. Anderson should be congratulated for presenting this case. It is the first one of its kind that I have encountered in nearly five years. Clinically I think the condition is blastomycosis. It would be interesting to find the organism in the histologic section.

DR. KENDAL FROST: I believe that this case of blastomycosis is the third I have encountered in Los Angeles in the last nineteen years.

DR. WILLIAM H. GOECKERMAN: Tuberculosis verrucosa cutis would be another guess, but the question will have to be studied further. The lesion is too dry for ordinary blastomycosis, one characteristic of which is the miliary abscess. An interesting feature of blastomycosis that I have wondered about is its comparative rarity in recent years. Some years ago there were many cases of blastomycosis in the Middle West. Dozens of them were observed. Suddenly the incidence lessened, and in later years there have been comparatively few. I wonder if any one has any information as to why that has happened.

DR. H. C. L. LINDSAY: I do not believe the condition is tuberculosis cutis. If it were, with such extensive areas of scarring, some new outbreak of tubercles would be found in the old scars.

Zosteriform Lichen Planus. Presented by DR. PAUL D. FOSTER.

A. S., a white man aged 20, came to see me in March 1939. At that time his condition was acute. He had vesicular lesions on the left side of the chest, extending to the mammary region, about the axilla and down the left arm. The grouping of the vesicles suggested herpes zoster. At the present time, instead of vesicular lesions, grouped yellowish papules are seen. The blood count and the urinalysis gave negative results. A serologic test for syphilis gave a negative result. Several intravenous injections of sodium iodide were given. About a week ago the treatment was changed to injections of mercuric salicylarsenate.

DISCUSSION

DR. SAMUEL AYRES JR.: I should prefer a diagnosis of linear lichenoid dermatitis. I do not think that the condition is lichen planus. The lesions are not the right color or the right shape. I cannot see any true lichenoid papules, and while linear, the eruption is not zosteriform.

DR. CHRIS HALLORAN: I do not observe any lichenoid lesions. Clinically I should favor the diagnosis of naevus unius lateris with a zosteriform arrangement. The patient also has lingua geographica.

DR. H. C. L. LINDSAY: One would expect lesions that have developed so rapidly to show some inflammation; microscopically there is but little.

DR. PAUL D. FOSTER: The diagnosis of zosteriform lichen planus was advocated because of the change in clinical appearance. The lesions appeared suddenly with what the patient described as "shooting pains." When first seen they were vesicular over the chest and around the axilla. In the intervening five weeks the clinical picture has changed somewhat, and although the lesions are not typical of lichen planus, they have assumed the distribution occasionally observed in lichen planus. The patient was receiving generalized ultraviolet irradiation because of his lowered vitality and the usual supportive measures in an attempt to build him up.

He came to the office originally with a geographic tongue of some twelve years' duration, but the lesions have disappeared entirely during the past three months with the stated regimen. I do not know what the diagnosis is. The condition began suddenly and with moderate suggestive symptoms. Certainly, had the patient been seen for the first time, the diagnosis of naevus unius lateris would have been made. The sudden onset suggested herpes zoster; later, as the vesicular appearance disappeared and a more flat, shiny appearance was assumed, the diagnosis lichen planus was considered.

A Case for Diagnosis (Papulonecrotic Tuberculid). Presented by DR. MAX WOLFF and DR. JOHN ROGERS (by invitation).

M. R., a white woman aged 62, presents a number of small grouped pitted scars on the extensor surfaces of the knees, elbows and ears. There is an active lesion on one ear about half the size of a dime with a depressed necrotic center. The present outbreak is of about four years' duration. Previous attacks date back about thirty years. It has been about ten years since a previous attack. An attack begins with erythematous swollen areas which are painful and tender. Many of the lesions appear as bullae, which later become pustular. There is a suggestion of bilateral symmetry. A serologic test for syphilis gave negative results, and a blood count was normal.

DISCUSSION

DR. H. C. L. LINDSAY: Papulonecrotic tuberculid is my diagnosis. I believe that if the patient were given a tuberculin test, if a careful study of serial sections were done or if an injection of some of the material were given to a guinea pig, the tuberculous nature of the disease might be confirmed.

DR. WILLIAM H. GOECKERMAN: This in my opinion is a classic papulonecrotic tuberculid. The classic distribution of grouped pitted scars over the extensor surfaces of the elbows and knees and on the lobes of both ears, the recurrent attacks in a reasonably healthy-looking person and the active lesion on the ear with a necrotic center all favor this diagnosis.

DR. SAMUEL AYRES JR.: I should consider a diagnosis of neurotic excoriations. The patient says that she picks the lesions and that they never heal until she digs the core out. The distribution certainly favors such a diagnosis. One might consider epidermolysis bullosa with secondary neurotic excoriations.

Acquired Progressive Kinking of Scalp Hair and Psoriasis. Presented by Dr. Samuel Ayres Jr.

R. N. W. is a white man aged 23. On the anterior portion of his scalp the hair is short, dry and wiry. The hair elsewhere is long and soft in texture. There is no change in the color of the hair. Scattered throughout the scalp are a number of small erythematosquamous plaques. There are similar patches on the chest and on the lower part of the back. This condition began five or six years ago. There are several typical patches of psoriasis on the scalp, chest and back.

DISCUSSION

Dr. J. R. Scholtz: I should like to ask Dr. Ayres if he has ever seen this condition in a man who was not beginning to show premature alopecia. It appears to me that I have seen this condition a number of times in young men with premature baldness and assumed that it was part of the senescence.

Dr. Samuel Ayres Jr.: This patient is only the second one with this condition whom I have seen. The other patient, I think, had lost a little more hair than this one; he came in for treatment of the scalp, because for two years he had noticed progressive kinking and an increase in pigmentation of the hair on the forward portion of the scalp. The patient now being presented had come in primarily for treatment of psoriasis; he had a few lesions on the scalp, a couple on the chest and one on his back. He had never had any treatment with roentgen rays on the scalp. Both the kinking of the hair and the psoriasis have been present for six years. When one looks at his scalp one becomes aware of the striking short, wiry, kinky appearance of the hair on the anterior portion, while the rest of the hair is soft and silky. At one time it was all of the same quality. There has been only one report of this condition that I am aware of, that of Wise and Sulzberger (Arch. Dermat. & Syph. **25**:99 [Jan.] 1932).

BRONX DERMATOLOGICAL SOCIETY

Samuel M. Peck, M.D., *President*

March 23, 1939

Henry Silver, M.D., *Secretary*

Epidermolysis Bullosa Acquisita. Presented by Dr. Charles Pines.

S. B., a woman aged 55, born in Poland, was seen on March 1, 1939, complaining of sores on the tongue, fingers and soles. The lesions appeared first about six months ago, on the buttocks, and healed spontaneously in a short time. Since then numerous vesicles and bullae have appeared on different parts of the body. The patient states that irritation or trauma preceded the appearance of many lesions on sites likely to be subjected to friction. No members of her family have ever been similarly affected.

There are three hemorrhagic bullae on the right border of the tongue, a ruptured bulla on the left buccal mucosa and a bulla on the left ring finger. On the extensor surfaces of the forearms and on the dorsa of the hands are numerous grouped milia-like cysts. Pigmentation and atrophy are present on both elbows and knees. On the soles there are a few ruptured bullae in the process of healing. The buttocks show areas of pigmentation, slight scarring and atrophic changes.

The coagulation time was five minutes. The sedimentation rate was 1.5 mm. in fifteen minutes, 5 mm. in thirty minutes, 8 mm. in forty-five minutes and 11 mm. in sixty minutes. Chemical examination of the blood showed 32 mg. of nonprotein nitrogen, 16 mg. of urea, 1.2 mg. of creatinine, 70 mg. of sugar and 9.2 mg. of

calcium per hundred cubic centimeters. The blood count revealed 82 per cent hemoglobin, with 5,000,000 red cells and 10,600 white cells per cubic millimeter. The differential count showed 45 per cent lymphocytes and 110,000 platelets. The Wassermann reaction was negative. Urinalysis revealed no abnormalities.

The histologic sections, examined by Dr. Timothy J. Riordan, showed a rather large bulla with separation of the epidermis at its junction with the corium. The underlying papillary layer of the corium was thinned, and the upper third showed clumping of the collagen fibers. Erythrocytes were seen in the space between. No noteworthy changes in the rete were observed. A section stained for elastic tissue showed destruction of elastic tissue fibers.

DISCUSSION

Dr. Moriz Oppenheim (by invitation): It is the first time that I have encountered a case of epidermolysis bullosa acquired late in life. One finds in the literature reports of such cases. Betmann distinguished between *Genodermie* and genodermatosis. The former is visible immediately after birth, as, for instance, epidermolysis bullosa hereditaria. Some such conditions are genodermatoses, in which event they make their appearance in later life. I do not doubt that the diagnosis of the presenter is correct, because the essential features of this disease are present. I have observed only the true hereditary type. A surprising feature in some cases is absence of the disease in the parents. It is known that epidermolysis bullosa hereditaria is one of the extreme dominant hereditary diseases. This is the reason why the case presented is so interesting. The hereditary factor in epidermolysis bullosa is recessive in the mother. Dr. Pines should investigate the patient's family more carefully. I recommend a trial of vitamin A, which is employed in the treatment of familial diseases.

Dr. Wilbert Sachs: The histologic structure is consistent with the diagnosis of epidermolysis bullosa. There was observed separation of the epidermis from the cutis with the papillary bodies still intact. There was no evidence of elastic tissue or remains of elastic tissue.

Dr. Frank E. Cross: I do not doubt that the condition is an acquired type of epidermolysis bullosa. It is my impression, however, that the disease is of much longer duration than the patient admits, especially because of the atrophic areas present.

Dr. Henry Silver: Attempts at therapy are discouraging. Administration of large doses of viosterol, as suggested by Ludy, of Philadelphia, may be tried.

Dr. Charles Greenhouse: I had occasion to treat a patient suffering from this type of epidermolysis. I used large doses of cod liver oil and exposure to sunlight, measures I had successfully employed in 2 recorded cases. Much to my surprise, there was great improvement. Of course, I do not know whether there will be a recurrence, but the patient has been well for about a year.

Dr. Samuel M. Peck: A synthetic vitamin A has recently been introduced. One is now able to get a preparation of almost any potency.

Dr. David Bloom: My impression is that treatment of epidermolysis bullosa is useless. If any improvement takes place after treatment it is due to a natural remission. I should like to inquire whether Dr. Oppenheim agrees with the term "acquisita" in this case, since there are many reported cases of this condition occurring in persons above the age of 40 or 50. I feel that in such cases, in spite of the late appearance of the lesions, the condition should be regarded as hereditary provided there is no indication for assuming a toxic cause, such as arsenic poisoning. The term "acquisita" should therefore be replaced by the term "tarda." The fact that no other members of the family are affected does not, of course, speak against the hereditary nature of the condition, for the dystrophic variety is known to be inherited as a recessive trait, while the simple form is inherited as a dominant trait. The condition in this case should be considered mildly dystrophic, for scars

are present. Many instances are found in the literature in which no other members of the family were affected.

DR. MORIZ OPPENHEIM (by invitation): "Acquired" epidermolysis bullosa is really not acquired, and the condition in Dr. Pines' case is of the true hereditary type. In true epidermolysis bullosa hereditaria, as the term is usually understood, the symptoms appear soon after birth or in the first year; but there are cases in which the lesions appear much later, and these are the cases of epidermolysis bullosa tarda, usually referred to as epidermolysis bullosa acquisita.

DR. CHARLES PINES: I inquired thoroughly into the family history, and I even questioned a few members of the family. To the best knowledge of the patient there is no one in the immediate or distant family similarly affected.

A Case for Diagnosis (Plantar and Palmar Keratosis with Dystrophy of the Nails? Psoriasis?). Presented by DR. SAMUEL FELDMAN.

I. S., a salesman aged 27, born in the United States, says that for as long as he can remember he has been annoyed with excessive sweating of the palms. About five years ago he noticed roughness of the skin of the right hand and beginning disfigurement of several nails. About one month later the condition extended to the left hand and to both feet.

The soles are red and hyperkeratotic. The redness and scaling extend to the sides and end in an abrupt line. Both palms are red and thickened. The natural lines are exaggerated. The redness extends to the backs of the fingers. The skin is moist from increased perspiration. The nails of the index, middle and ring fingers of the right hand are brittle, deformed and spoon shaped and present subungual keratosis. Slight pitting and transverse and longitudinal ridges are present on the remaining finger nails.

DISCUSSION

DR. MORIZ OPPENHEIM (by invitation): The patient presents two types of lesions: hyperhidrosis of the palms with koilonychia and fragility and hyperkeratosis of the soles with subungual keratoma. At first glance I thought of an occupational disease. It is known that workers in the dye industry, who use strong lotions for cleansing, show an enormous degree of hyperhidrosis. I have observed many such cases. One sees koilonychia also in metal workers and in persons handling dyes who use strong lotions for washing. There is no evidence of psoriasis either on the nails or on the skin. Moreover, hyperhidrosis is not a symptom of psoriasis vulgaris. One cannot bring both types of lesions under one heading unless one assumes a nervous disorder acting through the parasympathetic system.

DR. SAMUEL M. PECK: A condition such as is seen in this patient is not infrequent and in the last few years has been regarded as either psoriasis or dermatophytosis. However, I do not believe that the frequency with which the diagnosis of psoriasis is made necessarily means that that condition is present. Only too often such an eruption, in spite of the histologic picture, turns out to be a dermatophytosis. The fact that an eruption is localized on the hands and feet without psoriatic lesions elsewhere on the body speaks against a diagnosis of psoriasis. One must rule out from this group the bacterids. Andrews has called attention to this group in recent years. The histologic picture of this entity differs markedly from that of psoriasis. In fact, it more closely resembles that of the pustular dermatophytids. The condition is difficult to treat.

DR. HENRY SILVER: Koilonychia, as Dr. Oppenheim pointed out, occurs frequently in metal workers. I wish to call attention to another type of koilonychia, associated with gastric hyperacidity, the so-called koilonychia e stomacho.

DR. SAMUEL FELDMAN: The diagnosis of psoriasis or plantar keratosis was made tentatively. However, there is scaling on the side of the foot in the region of the great toe, suggesting psoriasis.

Acrodermatitis Continua Pustulosa (Hallopeau) Treated with Sulfapyridine. Presented by DR. MARION B. SULZBERGER.

Miss Y. S., aged 29, was presented at a meeting of the Philadelphia Dermatological Society (ARCH. DERMAT. & SYPH. **32**:815 [Nov.] 1935) and at a meetting of the Bronx Dermatological Society (ibid. **36**:443 [Aug.] 1937).

Prior to treatment with sulfapyridine there were acute lesions around the nails of the fourth and the great toe, on the sole along the metatarsal arch and on the lateral and posterior aspects of the right heel. The lesions in both groins were symmetrically arranged. On the right hand there were active lesions around the nails of the first, the third and the fourth finger and the middle portion of the phalanx of the third finger. On the left hand there were active lesions around the nails of the first and the fourth finger and on the hypothenar eminence. There were fissuring and crust formation on both lips, and the oral mucosa showed numerous denuded small areas and fissuring of the tongue. The occipital region of the scalp and the back of the neck were the sites of small and large confluent patches of somewhat fungating lesions.

At present there are three healing ulcerations in the occipital region as well as healing lesions around the nail of the left fourth finger. There are a few grayish red denuded areas on the oral mucous membranes and fissuring of the lips. The other sites have healed.

Examination of the blood on March 1, 1939, showed 80 per cent hemoglobin, with 8,650 white cells and 4,500,000 red cells per cubic millimeter. A differential count showed 39 per cent neutrophils, 32 per cent eosinophils, 1 per cent band forms, 3 per cent large lymphocytes, 24 per cent small lymphocytes and 1 per cent monocytes. Examination of the blood on March 22 showed 80 per cent hemoglobin, with 4,220,000 red cells and 12,750 white cells per cubic millimeter. A differential count showed 59 per cent neutrophils, 8 per cent eosinophils, 2 per cent basophils, 12 per cent large lymphocytes, 9 per cent small lymphocytes, 5 per cent monocytes and 5 per cent lymphoblasts.

During the preceding years the patient had been treated with staphylococcus toxoid, autogenous vaccine, streptococcus autovaccine, streptococcus antiserum, gold sodium thiosulfate, estrogen, silver arsphenamine, germanin, hyperpyrexia, vaccinia and carbon arc light, short wave and roentgen radiation, without benefit. A high caloric, high vitamin diet and different types of local treatment also had been given. She had received two courses of sulfanilamide without apparent benefit; the second course had to be discontinued because of vertigo, nausea and vomiting. From January 18 to February 15 the patient was given five intravenous injections of 1 per cent antimony and potassium tartrate U. S. P., but little improvement was noted. On March 1 she was given sulfapyridine, and she has now received 49 Gm. of the drug. Twenty-four hours after the beginning of this therapy an improvement was noted and has continued. However, while some of the lesions showed rapid improvement, others have shown themselves rather resistant to this therapy.

Histologic examination showed irregular acanthosis with hypergranulosis and hyperkeratosis. There were disassociation and vesicle and pustule formation in the epidermis. In the upper and middle layers of the cutis there were many dilated vessels, a number of newly formed capillaries and an infiltrate of small round cells, connective tissue cells and a few plasma cells. The histologic diagnosis was dermatitis vegetans, possibly pemphigus.

DISCUSSION

DR. I. BOTWINICK (by invitation): I wish to stress that twenty-four hours after treatment was instituted the result was amazing. The total amount of sulfapyridine given was 49 Gm. Repeated blood counts were made to watch the effect on the hemopoietic system.

DR. HENRY SILVER: Were examinations made to determine the concentration of the drug in the blood? I ask this because it has been recently demonstrated that

in spite of great variations in the concentration in the blood the clinical results are fairly constant.

DR. MORIZ OPPENHEIM (by invitation): I cannot say anything regarding the treatment, because I have had no experience with sulfapyridine. It would be indeed a miracle if such a result could be obtained in a case of acrodermatitis continua (Hallopeau). Treatment in the past has been so unsuccessful that I am somewhat skeptical regarding this therapy. I have always opposed the term acrodermatitis of Hallopeau because it is frequently confused with acrodermatitis atrophicans, and these two conditions have nothing in common.

It is my impression that Hallopeau's disease attacks all the fingers, as I had occasion to see at the international congresses in Paris, Copenhagen and Budapest. I am therefore in doubt whether this is a true case of Hallopeau's disease, in view of the fact that only a few fingers are involved. Also, the prompt response to therapy points to some infection, perhaps a dermatophytosis.

DR. SAMUEL M. PECK: The clinical picture which this patient presents would be a most unusual one for dermatophytosis. It is sometimes difficult to differentiate between pustular psoriasis and dermatophytosis.

DR. LOUIS CHARGIN: I see no evidence to warrant a diagnosis of pustular psoriasis. Hallopeau's disease is rather rare in the United States. One of the clinical features that should be stressed is that the disease begins with pustules around the finger nails. At times it spreads to the palms and the skin becomes undermined; it is then called dermatitis repens. I do not think that all the fingers necessarily are involved. It would appear that the condition of this patient is atypical Hallopeau's disease. The excellent response of the patient to sulfapyridine is perhaps due to the fact that Hallopeau's disease is, after all, a pustular disease. I may also state that it is regarded by some as a form of pemphigus. It is a matter of opinion. I do not feel, though, that it belongs in that group. Rather, I regard it as a pyogenic infection.

DR. DAVID BLOOM: I should like to be informed whether involvement of the groin, pubic region and scalp belongs to the picture of acrodermatitis continua. As far as I know, it does not. Streptococcus haemolyticus has been obtained on several occasions, and this would perhaps explain the curative action of sulfapyridine. My impression, like that of Dr. Chargin, is that the process is a pyogenic infection.

DR. PAUL GROSS: I agree with Dr. Oppenheim that it is difficult to define acrodermatitis continua of Hallopeau. Certainly, knowledge of pustular psoriasis and pustular bacterid (Andrews) has limited the diagnosis of acrodermatitis continua to a small number of cases. In a case which I observed there were a typical onset with paroncyhia and a slowly progressive border with small undermining pustules resembling impetigo herpetiformis. Although the condition was extremely chronic, it remained confined to one finger and one toe. The splendid results obtained in the patient presented seem to bear out the pyogenic nature of the disease.

DR. RUDOLF L. BAER (by invitation): With regard to the diagnosis, I should like to mention that Dr. Stokes, who saw the patient five years ago, regarded the condition as a form of pemphigus. When she was subsequently presented before this society, all agreed with the diagnosis of acrodermatitis continua of Hallopeau. In Philadelphia as well as here frequent cultures were made, but only nonpathogenic yeasts were found and on several occasions hemolytic streptococci. I think that the inefficacy of two courses of sulfanilamide shows that hemolytic streptococci were not the causative organisms. As far as the clinical picture is concerned, it is rather typical of acrodermatitis continua or dermatitis repens. The onset, location and progress all speak in favor of such a diagnosis. I think it would be unusual in dermatophytosis or psoriasis to find lesions of the mucous membrane or lesions of the groin of the type that this patient presents. Impetigo herpetiformis has been mentioned. This diagnosis was also considered, and studies of the blood calcium and phosphorus were made because of the possibility of hypo-

parathyroidism, which has been found to account for impetigo herpetiformis in most, if not all, cases. The blood picture was normal. In spite of this, however, the patient was treated with dihydrotachysterol, an irradiated ergosterol which has been successfully employed for eruptions due to hypoparathyroidism, including impetigo herpetiformis. No improvement was noted with this type of therapy. It was finally decided to try sulfapyridine, which produced the excellent result demonstrated.

Scrofuloderma. Presented by DR. SAMUEL FELDMAN.

D. S., a youth aged 19, a student, states that except for measles in early childhood he has always been in good health. About five years ago there developed a number of pustular lesions on the body. About three months ago a "boil" appeared on the inner side of the leg; it broke down and left an opening discharging pus. Soon afterward, a kidney-shaped lesion appeared on the outer side of the ankle and also exuded pus.

There is a fistulous tract about ½ inch (1.2 cm.) long situated about 2 inches (5 cm.) above the inner malleolus of the left leg. Thick, cheesy material was expressed from the opening on several occasions. Covering the outer malleolus there is a raised, soft, kidney-shaped bluish red lesion, from which creamy pus can be expressed.

Roentgenograms of the lung and of the left leg showed no abnormalities. Examination of the blood and of the urine gave negative results. The Mantoux test gave a positive reaction with a dilution of 1 : 1,000,000.

DISCUSSION

DR. LOUIS CHARGIN: The lesions on the ankle suggest tuberculosis verrucosa cutis rather than scrofuloderma.

DR. MORIZ OPPENHEIM (by invitation): I believe that this condition is not a case of scrofuloderma but rather a type of tuberculosis verrucosa cutis. I do not know whether I could differentiate on purely clinical grounds between tuberculosis verrucosa cutis and lupus verrucosus. In scrofuloderma there must be destruction of the lymphatic glands or of the superficial layers of the skin.

DR. WILLIAM LEIFER (by invitation): I wish to point out that the lesion over the achilles tendon was definitely adherent to it. The diagnosis of scrofuloderma therefore seems warranted.

DR. FRANK E. CROSS: The rather recent appearance of the lesions suggests the possibility of bromoderma. Has this been investigated? Can the diagnosis of tuberculosis still be mentioned in the absence of a fistula?

DR. SAMUEL FELDMAN: Bromoderma can easily be ruled out. There is a fistulous tract which leads to the tendon. The reason I regard the condition as scrofuloderma is that some assume two forms of scrofuloderma. One type is secondary to infection of the bones, muscles and glands, and the other may be primary and begins deep in the subcutis. The kidney-shaped lesion over the malleolus is not verrucous but may be readily mistaken for a verrucous lesion because of the rough surface and superimposed scales. It is a soft bag from which pus can be expressed.

A Case for Diagnosis (Lichen Planus? Lupus Erythematosus Disseminatus?). Presented by DR. FRANK E. CROSS.

V. H., a housewife aged 26, born in the West Indies, presents an eruption on the cheeks, arms, forearms and palms of five years' duration. On the right side of the face there are shiny violaceous papules forming circinate lesions about 3 cm. in diameter; a similar but smaller patch is seen on the left side of the face. Similar papular lesions are present on the flexor surfaces of both arms. There is general adenopathy. On both palms there are purpuric lesions the size of a pinhead. In 1932 the patient had an eruption diagnosed as secondary syphilis and

received fifteen intravenous injections at the City Hospital. She has received no further treatment.

Examination of the blood showed 65 per cent hemoglobin, with 2,900,000 red cells and 6,700 white cells per cubic millimeter. There were 58 per cent polymorphonuclears, 40 per cent lymphocytes and 2 per cent eosinophils and irregular cells. The Wassermann reaction was negative on two occasions.

DISCUSSION

DR. LOUIS CHARGIN: I favor the diagnosis of lupus erythematosus for the following reasons: The eruption is located on the face, in the favorite site for the disease, and it is of the superficial type. The eruption on the palms and fingers is that of disseminated lupus erythematosus. The sex of the patient is certainly not against the diagnosis. Lichen planus should be ruled out not because of the lack of typical lesions but because of the location and distribution of the eruption. Sarcoid may be considered, although the lesions of the face are not sarcoid, nor are lesions like those on the arms, which resemble erythema multiforme, observed in sarcoid. However, I think it is worth while to study the case further from that point of view. Of course, the enlargement of the glands favors sarcoid, but the leukopenia speaks for the disseminated type of lupus erythematosus.

DR. MORIZ OPPENHEIM (by invitation): It is difficult to make a definite diagnosis. My first impression was that of syphilis. However, serologic examination gave negative results, and for that reason this diagnosis can probably be excluded. In my opinion, the diagnosis can be made only by biopsy. Sarcoid must be considered because of metastases in the bones.

DR. WILLIAM LEIFER (by invitation): The patient presents generalized adenopathy. The epitrochlear glands are extraordinarily enlarged. This would favor Schaumann's disease.

DR. FRANK E. CROSS: While the distribution of the lesions is somewhat against lichen planus, the lesions are definitely lichenoid. Yet it must be admitted that many lesions are of the tuberculid type. I agree with Dr. Leifer's suggestion that this may be Schaumann's disease.

Lichen Sclerosus et Atrophicus. Presented by DR. MAURICE UMANSKY.

E. L., an obese woman aged 46, has had cutaneous lesions for the last three years. She has hypertensive cardiovascular disease of moderate severity and is suffering from polypoid ethmoiditis on the right and from pruritus of the genitalia due to nonspecific vulvitis of menopausal origin.

The eruption is located mostly on the left side of the abdomen (extending to the back in the lumbar region), on the sternum and on the flexor surfaces of the wrists. It consists of isolated lesions and groups of round or oval papules slightly raised above the surface of the skin. The papules are uniform, have an alabaster white center and a faint pink border and are the size of a millet seed or a split pea. In some areas of the abdomen they are grouped so closely that they appear to have formed a plaque; the individual lesions, however, are still discernible. Lately, scattered isolated papules have appeared on the upper part of the left side of the chest. There are no comedos or follicular plugs.

Examination of the blood showed 81 per cent hemoglobin. There were 10,200 leukocytes per cubic millimeter, with 62 per cent segmented polymorphonuclears, 2 per cent staff forms, 34 per cent lymphocytes, 1 per cent monocytes and 1 per cent eosinophils. The Kahn reaction was negative. The value for blood sugar was 125 mg. per hundred cubic centimeters of blood. Urinalysis showed a faint trace of albumin. The basal metabolic rate was + 6 per cent.

DISCUSSION

DR. SAMUEL FELDMAN: This is a case of typical lichen sclerosus. Most of the papules have evident pits in the center. On the shoulder there are numerous

red papules almost indistinguishable from lichen planus but with dilated follicles in the center. The diagnosis of lichen sclerosus can be made even on the basis of these lesions.

DR. CHARLES WOLF: The association of lichen planus with kraurosis vulvae is in my experience uncommon. I have treated a large number of patients with kraurosis vulvae, and in none was there any evidence of lichen planus or lichen sclerosus. A report has appeared (Hurst, E.: Leukoplakia Vulvae, Kraurosis Vulvae and Lichen Planus of the Vulva, *Brit. J. Dermat.* **48**:53-84 [Feb.] 1936) which stressed lichen planus as a precursor of kraurosis vulvae.

DR. HENRY SILVER: This is a typical case of lichen sclerosus. You will recall the case presented (Umansky, M.: ARCH. DERMAT. & SYPH. **39**:786 [April] 1939) a few months ago with a diagnosis of lichen sclerosus which I hesitated to accept because of the presence of white atrophic areas which were more characteristic of Csillag's disease. In this case, however, there are typical lesions of lichen sclerosus. There is also some suggestion of lichen planus, and I believe a biopsy may show a transition of lichen planus to lichen sclerosus.

DR. MORIZ OPPENHEIM (by invitation): Dermatitis atrophicans maculosa is a separate entity. Furthermore, I differentiate lichen sclerosus, scleroderma circumscriptum, morphea and white spot disease. White spot disease presents small lesions; morphea, lichen sclerosus and scleroderma circumscriptum show somewhat larger lesions. I separate lichen planus from lichen albus and lichen sclerosus. Lichen sclerosus and lichen albus are the same as Csillag's disease and Hallopeau's disease, but not lichen planus. Therefore, I have separated lichen albus and lichen sclerosus.

DR. SAMUEL FELDMAN: The main difficulty is the differentiation between Csillag's dermatitis lichenoides chronica atrophicans and the true atrophic forms of lichen planus. There is a primary atrophic lesion in the form of lichen planus sclerosus of Hallopeau, and there is a condition which I have described as lichen planus et acuminatus atrophicans. In my first article on that condition (*J. Cutan. Dis.* **37**:226 [April] 1919) I described the mode of transition of these atrophic lesions from lichen planus or from so-called lichen acuminatus papules (ARCH. DERMAT. & SYPH. **5**:102-213 [Jan.] 1922). Each begins with a definitely red papule, enlarges, becomes paler and finally becomes atrophic; the papules join to form atrophic patches in which every papule is still distinguishable. On the other hand, Csillag's disease begins as a white papule without a horny plug and does not resemble either lichen planus or lichen acuminatus. The papules join to form an atrophic dermatitis.

DR. MAURICE UMANSKY: The patient's eruption is uniform and presents one distinct type of lesion in all the affected areas. It does not show any follicular plugs, nor are diffuse atrophic plaques visible. These features are frequently encountered in cases of lichen sclerosus et atrophicus. Since the condition is extremely chronic and of many years' duration, one may miss some of the features at one time or another. The modern conception of lichen sclerosus et atrophicus considers the disease as a definite entity which occurs independently from lichen planus or scleroderma guttatum. The morphologic aspect of the eruption which the patient presents is so different from the aforementioned conditions that one has no difficulty in distinguishing them clinically. The uniformity of the lesions in this patient facilitates this differentiation. The various names under which cases of the identical type of eruption were described do not seem to be justified any longer. The clinical description of dermatitis lichenoides chronica atrophicans (Csillag's disease) corresponds to lichen sclerosus et atrophicus, and this condition should be placed in the same group.

Lichen Planus of the Mouth and Lips with a Peculiar Pigmentation of the Palate. Presented by DR. SAMUEL FELDMAN.

B. B., a housewife aged 45, noticed soreness in the mouth and blisters on the tongue about six weeks ago. Mastication became difficult, and she had to resort to soft food.

On the buccal mucosa there are grayish white glistening papules arranged in lines and uniting to form a mosaic network. On the dorsum and under surface of the tongue there are a number of grayish white patches. On the lips there are whitish lesions, and in several areas excoriations are present. The mucosa appears thinned, shiny and in some areas scaly. There are no lesions on the cutaneous surface of the body. The patient has been wearing a rubber plate for the past year. The portion of the palate covered by the plate appears normal. The area behind the plate, including the soft palate, presents decided yellow discoloration.

DISCUSSION

DR. FRANK E. CROSS: Such pigmentation is not uncommon in persons who wear rubber dental plates. It is probably due to the rubber, but I cannot explain the mechanism.

DR. ISADORE M. LASHINSKY: The yellowish pigmentation is located on the soft palate and is not at the site where the plate comes in contact with the palate. It is, therefore, difficult to incriminate the vulcanized rubber of the denture.

DR. SAMUEL FELDMAN: I examined this patient in daylight, and there is a definite yellow color of the mucous membrane which begins exactly where the hard palate ends. It is almost the color of fat.

DR. MORIZ OPPENHEIM (by invitation): I have observed several such cases. The pigmentation was associated with similar discoloration on the palms and was due to carotenemia from oranges, lemons and carrots. A Japanese first described it and stated that it was caused by the consumption of large quantities of oranges. This yellow tinge is also observed in young persons.

DR. LEO SPIEGEL: In the case of carotenoid pigmentation which I presented some months ago there was no evidence of discoloration of the mucous membranes.

DR. DAVID BLOOM: It is my impression also that the discolored plaque looks like that seen in carotenemia. However, this diagnosis can be considered only tentative, because the lesion is solitary.

A Case for Diagnosis (Inflammatory Papules or Deep Telangiectasia Occurring During the Course of Pregnancy). Presented by DR. SAMUEL FELDMAN.

Mrs. M. C., aged 30, was presented before this society at the November 1938 meeting (ARCH. DERMAT. & SYPH. **39**:784 [April] 1939).

She was delivered of a child in the beginning of January 1939. The baby died two hours after birth. Autopsy revealed hemorrhages in the brain and internal organs, notably in the kidneys and spleen. An emulsion was prepared from the placental blood and placental tissue. One cubic centimeter of this emulsion was injected into the mother intramuscularly twice a week. It was hoped that the emulsion would desensitize the mother against a possible toxin present in the fetal circulation and causing hemorrhages and death of the fetus and the palmar lesions in the mother. However, no changes were noticed in the lesions, and the red spots on both hypothenar eminences are as prominent now as when the patient was first seen.

DISCUSSION

DR. MORIZ OPPENHEIM (by invitation): This condition has been described as a congenital anomaly. I have seen 2 patients presented before the Austrian Dermatological Society in both of whom the condition was congenital.

DR. SAMUEL M. PECK: The clinical characteristics in this case offer decided differences from those of so-called Osler's disease, or hereditary familial telangiectasia. In the Osler syndrome the telangiectases are clearcut and have no surrounding erythema. In this patient one notes a zone of erythema and a sort of fuzziness to the margin of each of the lesions. This difference, I think, denotes that instead of a real nevoid type of telangiectasia there is a dilated vessel such

as is seen secondary to a toxic or an inflammatory process. This is more the type of telangiectasia which is seen in lupus erythematosus, erythema multiforme or the eruptions associated with sepsis. It is important in this case to test the capillary fragility with snake venom. In the nevoid type of capillary formation one gets a negative venom reaction; in the inflammatory type the reaction is often positive.

DR. PAUL GROSS: This is not a condition caused by pregnancy. I have observed it in a woman shortly after delivery as well as in a girl 7 years of age.

DR. SAMUEL FELDMAN: An interesting point in this case is that only during the three pregnancies which resulted in stillbirth did this condition occur. It did not appear during the pregnancy that resulted in a healthy child. The last baby died immediately after birth, and on autopsy hemorrhages in the internal organs were observed. There are no signs of syphilis and no history suggesting it. An endocrine disturbance must be considered.

Erythema Annulare Centrifugum (Darier). Presented by DR. LOUIS CHARGIN.

Mrs. P. G., aged 39, was presented before this society at the February 1932 meeting and subsequently at the November 1932 meeting (ARCH. DERMAT. & SYPH. **26**:577 [Sept.] 1932; **27**:886 [May] 1933).

She is represented now, seven years later, showing the same type of lesions which she had at that time. The lesions appear and disappear at irregular intervals. At no time, however, has the patient been entirely free of eruption. The lesions have the same papular, patchy character; many show circinate arrangement, producing bizarre figures enclosing healthy skin. At times there is severe itching. Recent examination of the blood gave negative results.

DISCUSSION

DR. MORIZ OPPENHEIM (by invitation): My first impression was that of mycosis fungoides. However, the long duration (nineteen years), the healing of the lesions in the center, leaving pigmentation, the seasonal disappearance of the eruption, the slight degree of itching, the absence of changes in the blood and the absence of characteristic histologic findings exclude mycosis fungoides. I suggest some form of erythema diutinum perstans configuratum. I do not believe it is parapsoriasis. The lesions are much too infiltrated and firm; some forms of erythema diutinum present firm lesions. Against this diagnosis is the desquamation.

DR. SAMUEL M. PECK: I think that it is pertinent at this time to bring up the question whether typical parapsoriasis ever becomes mycosis fungoides. Of course, one must assume that a correct diagnosis has been made in the first place. Has Dr. Oppenheim ever seen parapsoriasis en plaques change to mycosis fungoides?

DR. MORIZ OPPENHEIM (by invitation): In answer to Dr. Peck, I never saw true parapsoriasis turn into mycosis fungoides.

DR. PAUL GROSS: I did not fully agree with the conclusions drawn by Dr. Keil in his recent article (ARCH. DERMAT. & SYPH. **37**:465 [March] 1938). If strict criteria are applied, the diagnosis of parapsoriasis en plaques will be made less frequently, but there will still be a number of cases in which the findings will fit into the pattern of Brocq's disease. This is true particularly of cases in which the eruption starts in early adult life and shows the characteristic onset on the extremities and a gradual evolution of lesions on the trunk. The lack of any infiltration and the formation of fine scales as well as the uniformity of the plaques are other characteristics. On the other hand, I have no doubt that some eruptions diagnosed as parapsoriasis en plaques have turned out to be mycosis fungoides, despite the fact that the original diagnosis of parapsoriasis en plaques was made with reservations.

DR. SAMUEL M. PECK: The histologic picture of these lesions is doubtful. However, it has been my impression that study of the lesions of early mycosis fungoides enables one clinically to observe points of difference in the two conditions once the diagnosis has been established histologically.

DR. HENRY SILVER: I maintain that if one strictly adheres to the criteria given by Brocq fewer mistakes in diagnosis will be made, and consequently parapsoriasis en plaques will not eventuate in mycosis fungoides.

DR. CHARLES WOLF: I feel that this patient's eruption will terminate in mycosis fungoides. It bears strong resemblance in many respects to the one in the case presented before this society by Dr. Peck, in which there was an excellent response to roentgen therapy. In the past this patient had many recurrences, and only during the last attack could the diagnosis of mycosis fungoides be established.

In the early stages histologic study of such lesions furnishes little information.

DR. LOUIS CHARGIN: This patient has been free of lesions during long periods, sometimes for five to six months, without therapy. If roentgen therapy had been administered the improvement would have been attributed to that. The histologic picture is still not that of mycosis fungoides.

NOTE.—Histologic examination on March 28, 1939, showed that in the middle and upper layers of the cutis the vessels were dilated and there was cellular infiltrate, in part focal and in part diffuse. Throughout the middle and deeper layers of the cutis there was parenchymatous edema, but in the upper layers there was moderate interstitial edema. The cellular infiltrate was composed chiefly of small round cells and wandering connective tissue cells. There were no plasma cells or histiocytes (eosin–methylene blue stain). The epidermis was irregularly acanthotic, with considerable intercellular edema, spongiosis and vesiculation. Parakeratosis was present in several areas. The histologic diagnosis was dermatitis, or eczema.

Erythema Nodosum Associated with Vesiculobullous Lesions. Presented by DR. SAMUEL FELDMAN.

O. L., a housewife aged 39, was admitted to the Morrisania City Hospital on March 15, 1939, with painful lesions of the legs and forearms of ten days' duration. The condition began with an attack of grip accompanied by fever, nausea, vomiting and dryness of the throat. The patient stated that she had had two previous attacks, one of them severe enough to compel her to go to a hospital for treatment. The temperature was 100.6 F. on admission and on the following days ranged between 99 and 102 F. Examination of the blood showed 22,000 white blood cells per cubic millimeter, with 86 per cent polymorphonuclear leukocytes. Urinalysis gave negative results. The sedimentation rate of the erythrocytes was 30 mm. in sixty minutes.

On the anterior aspect of the legs there are large, bluish red, painful nodular lesions. They feel hot and are tender. Similar lesions are present on the extensor surfaces of the forearms. Some of these lesions show vesicles and bullae on the surface.

DISCUSSION

DR. CHARLES WOLF: It seems to me that the patient presents a drug eruption. My reason for this diagnosis is the definite history of recent grip for which a physician prescribed. Also, the lesions on the forearms, which are vesicular, are on an erythematous base. This suggests multiform erythema of drug origin.

DR. MORIZ OPPENHEIM (by invitation): One must consider secondary infection or perhaps a complication of erythema multiforme. I hesitate to make the diagnosis of erythema nodosum on the lesions now present, but the accompanying photographs definitely show lesions of erythema nodosum. One must therefore consider two conditions, erythema nodosum and a superadded multiform erythema due to an infection.

DR. CHARLES LERNER: I agree fully with Dr. Oppenheim. This patient had had erythema nodosum, and the vesicular lesions developed subsequently. Clinically there is no doubt that the diagnosis is erythema nodosum. On questioning the patient, I received the information that these vesicles developed after she had been given some medication for her sore throat and what she called "grip." The medicament was in all probability a coal tar product.

DR. SAMUEL FELDMAN: When I first saw the patient she presented lesions of erythema nodosum with accompanying symptoms, elevation of temperature and pain. The lesions still are painful and tender. On the tenth day of the disease a vesicle developed on one lesion, and subsequently a few more appeared. The eruption is probably not due to a drug, because the patient took only one capsule, and the vesicles developed about ten days later.

NEW YORK DERMATOLOGICAL SOCIETY

J. FRANK FRASER, M.D., *Chairman*

March 28, 1939

FRANK C. COMBES, M.D., *Secretary*

Neurogenic Sarcoma of the Skin. Presented by DR. J. FRANK FRASER.

V. M., a man aged 40, of Italian parentage, presented himself at the Skin and Cancer Unit with a walnut-sized nodule on the right cheek near the margin of the upper lip. The lesion first appeared four years ago, at which time it was removed surgically and the site was treated with roentgen rays (the dose is unknown). The growth promptly recurred and was again removed, but it reappeared at the margin of the original site a year after the second operation.

The results of laboratory tests were negative. Microscopic examination showed the histologic picture of fibrosarcoma of neurogenic origin.

DISCUSSION

DR. EDWARD R. MALONEY: In view of the pathologic report presented by the chairman, I think the diagnosis made must be accepted.

DR. GEORGE M. MACKEE: Clinically the growth might be an epithelioma. Histologically, of course, it is a fibrosarcoma, or what some call a spindle cell sarcoma. Spindle cell sarcoma is exceedingly difficult to eradicate, because it is so difficult to tell where the diseased tissue ends and the normal tissue begins. Recurrence is common. The excision must be extremely wide and deep. Fortunately, these tumors are not of a high degree of malignancy; so the prognosis for life is not hopeless. The tumors are not radiosensitive. I favor exceedingly wide excision, so wide that a plastic operation may be necessary.

Prickle Cell Epithelioma of the Scrotum. Presented by DR. FRED WISE.

J. V. H., a clerk aged 56, presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, has a tumor on the inferior surface of the scrotum, said to be of one and one-half years' duration. The lesion is irregularly oval, 4 cm. long and 1.5 cm. wide, and consists partly of an elevated pea-sized nodule situated on a flat, plateau-like, sharply defined yellowish pink plaque. There is no palpable adenopathy in the crural regions, and subjective symptoms are absent. The patient states that he has never come in contact with such materials as paraffin, soot or tars.

DISCUSSION

DR. GEORGE M. MACKEE: I agree with the clinical diagnosis of squamous cell rather than basal cell epithelioma. It is probably a grade 1 epithelioma.

DR. EUGENE F. TRAUB: I appreciate that the usual type of epithelioma found on the scrotum is the squamous cell type, but the long duration in this case suggests that the growth might conceivably have started as a basal cell lesion and the one portion of it which is slightly more infiltrated than the rest might show a squamous cell involvement. Naturally it is possible that the entire lesion may simply be a slowly growing squamous cell epithelioma. The clinical appearance of the lesion, if seen for example on the face, would certainly suggest a basal cell type rather than a squamous cell type to me.

DR. J. FRANK FRASER: I disagree with the view that this is a basal cell carcinoma. The clinical features are typical of a squamous cell tumor of low grade malignancy, probably grade 1 (Broder).

DR. M. OPPENHEIM (by invitation): The tumor is like the chimney sweep epithelioma in England. I agree with Dr. Fraser and Dr. MacKee that clinically this is a prickle cell epithelioma. It is not due to the patient's occupation. In occupational cancer are seen warts, thickening of the skin, hyperkeratoses and other changes in the skin, and when occupational cancer is observed it is always ten or fifteen years after the patient has worked. I believe that the tumor in this case is a prickle cell epithelioma, which has been observed in other places on the skin, but it may be that the scratching of the scrotum has something to do with its development.

DR. J. FRANK FRASER: I agree with Dr. Oppenheim's view that the appearance is suggestive of occupational cancer but that the duration is too short. Occupational tar cancer, for example, usually requires constant contact for one fifth of the span of life. This rule holds good both in human beings and in the lower animals.

DR. EUGENE F. TRAUB: Apparently I have been misunderstood. I realize that a basal cell epithelioma on the scrotum would be an exceptional lesion, as most epitheliomas in this location are of the squamous cell type. The point I tried to make was that it is the location of the lesion rather than the clinical appearance which guides one in calling this lesion squamous cell epithelioma, because, as I stated before, a lesion like this on the face would be more correctly called a basal cell type.

DR. GEORGE M. MACKEE: I do not agree with Dr. Traub. I will admit, of course, that one cannot absolutely differentiate clinically between the basal cell, the squamous cell and the basosquamous cell type in any location other than on the mucous membranes. There is nothing in this case that would warrant a diagnosis of basal cell epithelioma except perhaps the duration. There are no pearly nodules, rolled edges or destructive ulceration. The lesion is a slowly growing, hard, opaque mass, which is more characteristic of squamous cell than of basal cell epithelioma.

Lupus Erythematosus Disseminatus Chronicus. Presented by DR. FRED WISE.

S. T., a girl aged 18, was presented by Dr. Max Scheer at the meeting of the Manhattan Dermatological Society on March 14, 1939.

DISCUSSION

DR. M. OPPENHEIM (by invitation): This is certainly a case of chronic discoid lupus erythematosus with pigmentation. Reports of some cases of this type have been published with a diagnosis of poikiloderma. I believe that most examples of Civatte's type resemble this type of lupus erythematosus pigmentosus discoides chronicus.

DR. GEORGE M. MACKEE: The condition is probably the disseminate type of the disease. The patient has not responded well to treatment with bismuth and gold preparations. Treatment with sulfanilamide might be successful. The unusual pigmentation may be the result of the gold and bismuth therapy.

DR. EUGENE F. TRAUB: I should like to hear something about the prognosis. Is the disease liable to be fatal?

DR. FRED WISE: The object of presentation was chiefly to show the pigmentation and the complete loss of eyebrows due to atrophy of the skin. The patient showed pronounced atrophy of the skin and admitted picking the scales from her nose, producing a verrucous appearance of the nose, which is not a natural sequel of lupus erythematosus, treated or untreated. She states that there has been no improvement after fifty injections of a gold compound, but I think there has been some improvement in the active process itself, though none in the intensity of the hyperpigmentation. In answer to Dr. Traub's question, I do not think this is the type of case in which the prognosis as to life is bad, but as regards the cosmetic defect, I think the prognosis is poor. I do not think the cosmetic defect can be easily remedied by any means. I am unwilling to agree with Dr. Oppenheim's views regarding the resemblance of the eruption to poikiloderma of Civatte.

Atrophia Cutis Maculosa. Presented by DR. FRED WISE.

A. M., a woman aged 48, on March 23, 1939, presented an eruption of fifteen years' duration on the back, chest, abdomen, arms and thighs. The entire torso presents numerous closely aggregated macules, varying in diameter from 0.5 to 1 cm. Similar but less numerous macules are on the arms, shoulders and thighs. The lesions are reddish brown and smooth, and some are slightly depressed below the surrounding normal skin. The general appearance is similar to the sequelae of a healed eruption of generalized lichen planus. There is no history of ingestion of drugs.

NOTE.—In a subsequent report Dr. Satenstein stated that histologic study of a lesion from the chest revealed absence of elastic tissue in the upper part of the cutis, which suggests the diagnosis of primary macular atrophy.

DISCUSSION

DR. GEORGE M. MACKEE: The patient was questioned carefully, and a history of an antecedent eruption could not be elicited. My diagnosis is evasive and does not indicate a definite entity, but I should call the condition primary macular atrophy. It is the only case of primary macular atrophy that I have observed associated with pigmentation instead of with erythema.

DR. EDWARD R. MALONEY: I agree with the diagnosis made by Dr. Wise.

DR. M. OPPENHEIM (by invitation): I think that this is not the condition described by Jadassohn, which is now called dermatitis atrophicans maculosa (anetoderma), because in this condition are three stages, of which the first is a circumscribed area of erythema; the second is that of depigmentation, in which a ring forms which has a violet-red zone at the periphery and a pale center, and in the third stage there is atrophy, so the skin on palpation feels as if there were a hole in it. There is no doubt that there is atrophy in the case under discussion, and there is no doubt that there is pigmentation, but I believe there is secondary macular atrophy. The history is negative in this regard, but the differences in the size of the macules and the too regular pigmentation, with the absence of baglike depressions with cigaret-paper-like wrinkling, makes me feel that the condition belongs to the secondary macular atrophy group described by Balzer and other authors. I should like to hear the opinion of Dr. Wise.

DR. FRED WISE: I agreed with what Dr. Oppenheim has said, especially in regard to two points. One is that I should not consider the diagnosis of Jadassohn's anetoderma of the disseminated macular type of atrophy, in which there are, as he said, elevated, baglike lesions. The second point in which I agree is that the

history is misleading. The patient probably does not remember what happened fifteen years ago, and she probably did have a preceding eruption. What was that preceding eruption? Was it lichen planus? That is the point I wish to bring out.

Xeroderma Pigmentosum. Presented by Dr. Joseph J. Eller.

A. J., a boy aged 18 months, was first seen in May 1938, at the age of 9 months. He had an intertriginous eruption in the anogenital region and in the groins. He also had numerous small pigmented macules on the face. The skin in these areas was hyperkeratotic and desquamating. The mother stated that this condition had been present for about one month. The general health was good.

An older brother also had xeroderma pigmentosum in which basal cell and squamous cell carcinoma appeared. Metastases developed, and he died at the age of 5 years. The mother and father are Russian Jews. One sister is 14 years of age and is physically and mentally normal.

Despite all efforts to avoid exposure to sunlight and despite the constant local application of emollients, the xeroderma pigmentosum in this patient has steadily progressed. More pigmented macules have appeared, and small keratoses have developed.

DISCUSSION

(The diagnosis was accepted unanimously.)

A Case for Diagnosis (Duhring's Disease? Pemphigus?). Presented by Dr. Fred Wise.

E. M., a woman aged 49, registered at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on March 21, 1939, with an eruption on the arms, chest, neck and face of five years' duration. The patient states that the eruption is always exacerbated by acetylsalicylic acid. Itching is not prominent. The lesions vary from erythematous macules to large raised, shallow encrusted ulcers. There are scattered pustules and vesicles, the latter appearing to be grouped in arcs. One large bleb is seen under the lobe of the right ear.

The patient is now under treatment for diabetes mellitus with insulin. Two weeks ago an attack of grip with a sore throat was followed by cervical adenitis on the right side. The condition is now subsiding.

Patch tests with potassium iodide and potassium bromide gave negative results. The Wassermann and Kahn reactions were negative. Examination of the blood showed eosinophilia, with 3 per cent eosinophils, but otherwise gave negative results.

Note.—A subsequent histologic diagnosis of dermatitis herpetiformis was made by Dr. Satenstein.

DISCUSSION

Dr. R. H. Rulison: The case impressed me as being one of self-produced eruption.

Dr. Eugene F. Traub: I believe the condition might be a dermatitis medicamentosa due to iodides or bromides. Has this diagnosis been absolutely excluded?

Dr. Frank C. Combes: I do not agree with the diagnosis of dermatitis herpetiformis for several reasons. In the first place, the eruption is too localized. Although at times this disease may occur in one isolated area, I think it is extremely difficult to make a diagnosis in such cases. Furthermore, there is an entire lack of pigmentation and of characteristic excoriations. The history is unusual. There have been no remissions. On the contrary the eruption has persisted for a long time with gradual extension. Finally, such an extensive erythematous eruption of the face I have never seen in Duhring's disease. I should like to suggest the diagnosis of pemphigus erythematodes.

DR. HOWARD FOX: I agree with Dr. Combes that pemphigus erythematodes should be considered, although there are no lesions on the back, which is usually involved in cases of this disease. I do not think this is a factitious eruption, as it is entirely symmetric. In favor of a diagnosis of atypical Duhring's disease is the undoubted presence of excoriations, in spite of the fact that the patient said she had not scratched the lesions. Furthermore, there is at least one unruptured vesicle on the lip.

DR. EDWARD R. MALONEY: I agree with what Dr. Combes said about this not being a case of dermatitis herpetiformis for the reasons he advanced. My impression is that it is a case of neurotic excoriations. It seems to me that most of these lesions, or at least a great many of them, are excoriated.

DR. GEORGE M. MACKEE: When I first saw the patient, at a distance of perhaps 15 feet (4.5 meters), I thought because of the excoriations and the distribution that she had neurotic excoriations, but then I noticed that the elementary lesion is a vesicle which appears in unaltered skin, after which the surrounding skin becomes erythematous. Some of these elementary lesions are discrete and some grouped, and sometimes they form a configuration. I thought of dermatitis herpetiformis and pemphigus and finally came to the conclusion that the condition might represent the Senear-Usher syndrome. There is no history of ingestion of drugs. I think she said she had taken acetylsalicylic acid, with a flare-up of the eruption afterward, but she does not take this medicament frequently.

DR. HOWARD FOX: As acetylsalicylic acid was mentioned as a possible cause, I should like to ask if any one present has seen an eruption from that drug approaching the extent of this one. In view of the fact that the drug is used so extensively, I doubt whether more than an occasional trivial eruption is ever caused by it. I personally have had an occasional slight morbilliform rash following the use of the drug.

DR. FRED WISE: In considering the question of a drug eruption, I believe that the history of the patient must be taken into consideration. This patient says that she has not taken any drugs except occasional doses of acetylsalicylic acid, and she has had the eruption without taking this drug over a period of several months. Therefore, I am inclined to eliminate the possibility of a drug eruption. The eruptions due to acetylsalicylic acid, of which I have observed only 2, have been morbilliform and erythematous and have not resembled this one. I do not think the eruption resembles any form of pemphigus; to me it resembles an aberrant form of Duhring's disease.

Psoriasis Amiantacea. Presented by DR. FRED WISE.

J. C., an Italian aged 60, registered at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on March 28, 1939. His eruption is of seven years' duration and appears, with remissions and exacerbations, on the extremities. The eruption is confined to the backs of the hands, the elbows and the skin of the feet and ankles. On the hands and elbows, the lesions consist of well defined discoid and oval hyperkeratotic lesions, ranging from 1 to 4 cm. in diameter, on which the scales can be readily detached. Over the internal and external malleoli of both ankles the discoid lesions have sharply defined outlines, are approximately 4 cm. in diameter and present a dirty grayish nonscaly granular appearance, similar to that of asbestos. These plaques are resistant to topical remedies and have not changed for several years.

NOTE.—Subsequent histologic study of specimens from the lesions of the elbow and ankle by Dr. Satenstein confirmed the diagnosis of psoriasis.

DISCUSSION

DR. HOWARD FOX: I do not think any part of the eruption is characteristic of psoriasis. There is no family history of this disease, and there are no changes in the scalp or nails to suggest psoriasis. I should call this a chronic lichenified

eczema with an unusual amount of hyperplasia of the horny layer on the malleolus. I think the term amiantacea, meaning asbestoslike, is not suitable in this case.

DR. FRANK C. COMBES: I agree with Dr. Fox. If the patient had not had the slightly scaly patches on the elbows, I doubt if a diagnosis of psoriasis would have been entertained. However, the lesions on the elbows have scales which are not micaceous but look to me like lichenification or lichen simplex circumscriptus.

DR. GEORGE M. MACKEE: I should not definitely rule out psoriasis as the diagnosis in this case, because psoriasis can produce some unusual manifestations. The lesions on the elbows to some extent suggest psoriasis, and even those on the backs of the hands and the large patch on the ankle could, I think, be evidence of psoriasis. In an informal discussion Dr. Oppenheim mentioned the possibility of keratosis verrucosa, which is really a variety of neurodermatitis. The final diagnosis in some of these cases of circumscribed neurodermatitis, after careful study, is amyloidosis or lipoidosis.

DR. M. OPPENHEIM (by invitation): As Dr. MacKee has said, I had the idea that this might be the disease which was described by Weidenfeld under the name keratosis verrucosa, which belongs to the lichen simplex group. The eruption here is symmetric, but that of psoriasis is not. It is surprising that the patient has patches on the knuckles, on the elbows and on the ankles, but I do not believe the condition is psoriasis. The histologic picture may be that of psoriasis, but clinically the condition resembles a neurodermatitis with secondary lichenification. It may be due to some reaction of the tissues in the form of amyloidosis or lipoidosis. The absence of itching is important. From the clinical aspect alone, I am led to the diagnosis of neurodermatitis verrucosa, an old verrucous form of neurodermatitis chronica.

A Case for Diagnosis (Onychitis and Occupational Dermatitis? Fungous Infection?). Presented by DR. FRED WISE.

G. B., a man aged 54, registered at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on March 28, 1939, with an eruption confined to the backs of the hands, the finger tips and the finger nails. A similar eruption first appeared when he was 14, persisted for seven years and did not recur for twenty-three years. The present eruption appeared eight weeks ago. The patient states that the eruption disappears when he stops work. He is a porter and handles metal polish and soaps and has his hands in water most of the day.

The finger tips present a severe form of erythematous, denuded dermatitis with a glistening appearance. The nails are thickened, discolored and friable.

NOTE.—The results of mycologic examinations of both hands and feet were negative.

DISCUSSION

DR. GEORGE M. MACKEE: The condition seems to be either dermatophytosis or dermatitis venenata.

DR. M. OPPENHEIM (by invitation): I do not believe this condition is a contact or occupational dermatitis. The lesions between the fingers have some resemblance to those in monilia infection, as Dr. Hopkins has described it. Diagnosis is possible only by microscopic and cultural examinations. The eruption is sharply limited and absent on the fingers, and the subungual swelling is associated with a nearly normal appearance of the nails, which is not observed in occupational dermatitis.

DR. EDWARD R. MALONEY: I agree with the previous speakers and feel that this case should be investigated for a possible fungous infection.

DR. FRED WISE: I agree with the previous speakers as to the likelihood of a fungous infection but I have no doubt whatever that the condition is also an occupational disease, in addition. The patient says that if he stops working for

three days the eruption clears up. It might be a monilia infection or dermatophytosis and still be an occupational dermatitis. He handles cleaning fluids and polishes and thereby aggravates the preexisting infection.

Cavernous Hemolymphangioma of the Tongue. Presented by DR. FRED WISE.

L. L., a man aged 64, a patient of Dr. Charles Rein, has a lesion on the left half of the tongue which has existed since birth. On the tongue is a sharply marginated, diffuse swelling, involving the distal two thirds of the left half of the tongue and extending slightly beyond the midline. The involved area is soft and spongy and shows a mottled red and blue discoloration. There are no ulcerations.

The patient never suffered any subjective symptoms until one week ago, when he had eight teeth extracted. Decided swelling of the left side of the face developed immediately. There are still some swelling and cervical adenopathy.

DISCUSSION

(The members accepted unanimously the diagnosis presented.)

A Case for Diagnosis (Sporotrichosis?). Presented by DR. EDWARD R. MALONEY.

C. de S., a man aged 48, born in Italy, employed as a porter in a hospital for contagious diseases, presents an eruption consisting of several deep seated nodules on the surface of the right forearm, almost in a line. The initial nodule appeared at the base of the right thumb three months ago, and the others followed in succession, the last one developing ten days ago. His temperature and pulse have been normal, and he has had no particular subjective symptoms. He has a positive Wassermann reaction but says he has not had syphilis. The reaction to a tuberculin test was negative. The results of cultures and of several inoculations in animals have been negative. Histologic examination of tissue from one of the nodules showed the structure of a granuloma with a few foreign body multinucleated giant cells.

DISCUSSION

DR. HOWARD FOX: From the few cases I have observed and the numerous illustrations in the literature, I should make a clinical diagnosis of sporotrichosis. Tularemia might be a possibility except that there have been no febrile symptoms and no history of contact with rabbits. Dr. Maloney has investigated this case carefully, and it is possible that further study may confirm the clinical diagnosis.

DR. FRANK C. COMBES: I do not know of any other condition which this simulates more than it does sporotrichosis.

DR. EUGENE F. TRAUB: I agree that sporotrichosis is the best diagnosis. It remains to be proved.

DR. R. H. RULISON: I think the condition will eventually prove to be sporotrichosis.

DR. J. GARDNER HOPKINS: The lesions on the arm are in the skin and not in the subcutaneous lymph nodes, which eliminates the diagnosis of tularemia. I do not understand why the diagnosis should not be sporotrichosis except that Dr. Maloney has not been able to prove that it is. It is hard to believe that the organism did not grow if the tissue for culture was taken from the depth of the lesions. I wonder if the linear distribution might not be fortuitous and if the dermatitis might not be due to the patient's occupation or be a bromoderma or iododerma.

DR. GEORGE M. MACKEE: Dr. Maloney has worked up the case well and the histologic examination shows nothing significant, which certainly ought to rule out, for instance, the possibility of syphilis and tuberculosis. The evolution is too rapid for tuberculosis. Against a diagnosis of sporotrichosis is the fact that these

lesions are superficial. They appear to be in the skin, while the lesions of sporotrichosis are usually, I think, subcutaneous in the beginning. However, the linear distribution of nodules deep in the skin along the forearm, following an initial injury and lesion of the hand, makes the diagnosis of sporotrichosis probable.

DR. EDWARD R. MALONEY: The nodules were deep.

DR. M. OPPENHEIM (by invitation): I agree with the diagnosis of sporotrichosis. The lesions are distributed along the lymphatics and are deep and fluctuant.

DR. EDWARD R. MALONEY: Of course the diagnosis of sporotrichosis is still possible in spite of the fact that it has not been proved. The lesions are deep in the skin and not superficial. The histologic structure is that of a granuloma. There are cells which suggest foreign body giant cells. I do not know of any other diagnosis that can be made as readily as sporotrichosis.

Amyloidosis Cutis Lichenoides. Presented by DR. R. H. RULISON.

T. P., an Italian aged 72, was presented by Dr. Charles A. Greenhouse before the Bronx Dermatological Society with the diagnosis of lichen planus ocreaformis in 1935 (ARCH. DERMAT. & SYPH. **32**:513 [Sept.] 1935) and also referred to in the discussion of another case (ARCH. DERMAT. & SYPH. **35**:1168 [June] 1937).

The patient is now presented from the Dermatological Clinic of Lincoln Hospital. He was formerly employed in the building trades but is at present unemployed. Seven years ago he had a mastoidectomy on the right side. Except for his present condition he has had no illness.

Thirteen years ago intense itching developed in both legs above the ankles. The itching lasted ten years and ceased three years ago. An eruption developed and eventually involved both legs symmetrically, from 2 inches (5 cm.) above the ankles to the junctions of the upper and the middle thirds of the legs. As described in the previous presentation, "Scratching to relieve itching causes vesicles to appear in the affected areas. There are also some exudation and formation of crusts. The itching ceases when the blisters open. For the last four or five years the condition has remained stationary." At that time the eruption was described as "composed of numerous discrete purplish red papules about the size of a lentil. There is considerable pigmentation."

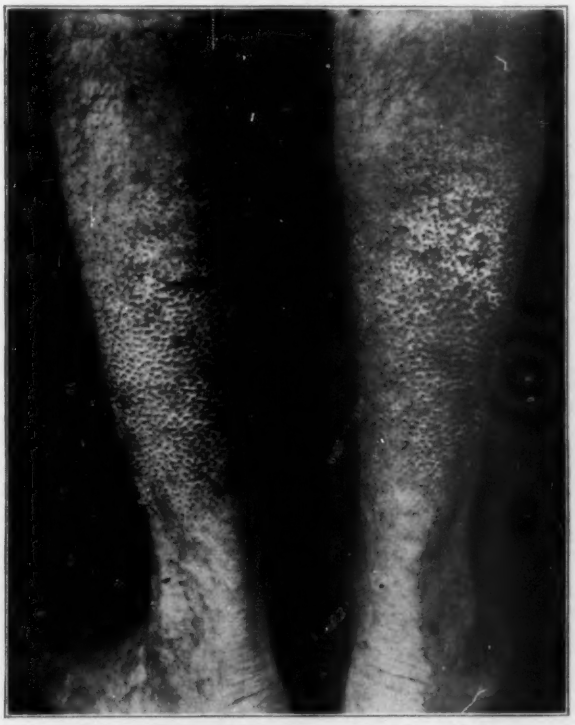
For the past three years there has been no itching. No vesicles or bullae have occurred. There is no evidence of scratching in the affected areas. However, the patient complains of intense itching on the outer surface of the left thigh, and examination shows numerous scratched areas on other parts of the body. The elbows and knees show dry hyperkeratotic scaling. The skin is normal except on the legs and elbows.

As for the present appearance of the legs, from the junction of the upper and the middle thirds of the legs to the line of the shoe tops there is a uniform, closely aggregated but discrete collection of slightly brownish, sugar-loaf–shaped hard papules. These are somewhat more elevated on the flexor surfaces than over the shins. They are uniform in size, having the diameter of a match head or a millet seed. The tops of these papules are smooth and glistening. The clefts between them show fine white scaling. None of the papules show polygonal outlines, as found in lichen planus. The skin feels thickened, and the general appearance is pebbled.

Dr. Greenhouse stated, "In my case injections of congo red gave absolutely negative results." It must be assumed that this test was made by intravenous injection and was used to demonstrate the presence or absence of generalized amyloidosis. Nomland stated that this form of injection does not stain amyloidosis of the skin. The point is of interest because the test presumably shows that this patient is not suffering from the generalized type of the disease. The long duration of the local condition and his general good health confirm this.

On March 23, five days ago, 1 cc. of a 1 per cent solution of congo red was injected subcutaneously on the inner side of the left calf. At the same time 0.1 cc. the same solution was injected intradermally into each of the two neighboring areas. The characteristic absorption of the stain into the areas of amyloidosis is to be seen in an area the size of the palm. The papules in this area now are bright red.

The patient is in good health. His only complaint is itching, which is most annoying on the left thigh anteriorly and laterally. From examination it is evident that he has generalized pruritus but none on the legs. His pulse rate is 92. His blood pressure is 140 systolic and 90 diastolic. The heart sounds are distant, and there are no murmurs. The heart is not enlarged. The lungs are normal.



Amyloidosis cutis lichenoides.

The abdomen is soft, and no masses are felt. The liver and spleen are not palpable. The eyegrounds are normal. There are no pathologic reflexes, such as the Babinski or Gordon, and no clonus. Knee jerks are slightly hyperactive and equal. The Wassermann reaction was negative. Examination of the urine for Bence Jones albumose gave negative results. The tongue and the buccal mucosa are normal.

The patient was first seen at the Lincoln Hospital on Sept. 11, 1933. A diagnosis of lichen planus hypertrophicus was made. Later this was changed to lichen planus ocreaformis. He received injections of a bismuth preparation and solution of potassium arsenite U. S. P. (Fowler's solution) and local applications. Biopsy specimens were taken on Nov. 15, 1934, in 1935 and also on Nov. 21, 1936. Reexamination of these specimens and special staining were done. Dr. A. Liber, of Lincoln Hospital, made the following report:

"In the specimen taken on Nov. 15, 1934, there was moderate hyperkeratosis, with scaling and moderate keratosis pilaris. The stratum granulosum was well preserved and broadened over the central portion of the lesion except at one point, where there was incipient parakeratosis. The prickle cells in this region were swollen, and many presented different degrees of spongiosis. There was marked acanthosis with long rete pegs of irregular shape. The papillae of the derma were swollen and drumstick shaped. In the center of the lesion the papillae and the rete were fibrosed and contained masses of homogeneous pink-staining material, the tinctorial affinities of which were somewhat different from those of collagen, although they sometimes appeared identical in form and position with the neighboring thick collagen bundles. In these regions the blood vessels were dilated. A large hair follicle ran through the center of the lesion. The rete of the neighboring portions of the skin contained nodular infiltrates of lymphocytes and histiocytes. The swelling of the papillae faded out gradually toward the normal skin, which presented melanosis of the basal layer. In the central portion were small, irregular clefts between the epidermis and the derma. They contained faintly pink-staining material. It was difficult to determine whether they were artefacts or preexisting.

"Later biopsy specimens presented a similar picture but showed increasing keratosis pilaris and acumination of the epidermal crests. With methyl violet the homogeneous material stained red, but it remained unstained with iodine solution, both with and without sulfuric acid. The congo red stain was ineffective.

"Conclusion: The material deposited in the cutis was atypical amyloid."

The original slides, together with recently stained ones from the same blocks, as well as photographs of the legs are submitted. It is interesting to compare these photographs with those in the article by Nomland (ARCH. DERMAT. & SYPH. **33**:86 [Jan.] 1936), the illustration of lichen planus ocreaformis found in Sutton's book (Diseases of the Skin, ed. 7, St. Louis, C. V. Mosby Company, 1928, p. 192) and the one in Winer's article (ARCH. DERMAT. & SYPH. **23**:870 [May] 1931).

An interesting point is that there was itching of the involved area for ten years and there has been none for the past three years.

NOTE.—The patient was hospitalized, but no significant observations were made. Tests with congo red injected intravenously showed no retention or absorption of the dye.

DISCUSSION

DR. J. GARDNER HOPKINS: I feel instructed by having observed this case. It is extremely interesting. There is not much left to discuss.

DR. EUGENE F. TRAUB: I agree with Dr. Hopkins.

DR. HOWARD FOX: Dr. Rulison deserves great credit for proving the presence of localized amyloidosis as shown by the presence of the red color following localized injections of congo red. I should like to hear Dr. Rulison's opinion about the well known case of Dr. Liebenthal, in which a diagnosis of lichen planus ocreaformis was made.

DR. M. OPPENHEIM (by invitation): I have never seen amyloidosis circumscripta with such a wide extent, covering nearly the whole surface of both legs. Both types have been observed, the generalized amyloidosis, which is different from this, and the local form. However, I have seen it stated in the ARCHIVES that lichen planus is the initial stage of the condition. Does Dr. Rulison too believe this? Amyloidosis circumscripta is observed in cases of lichenified skin. The condition in these cases impresses me as lichen simplex and not lichen planus. What is the opinion of the members?

DR. GEORGE M. MACKEE: There is a feature that is rare in this disease. A patient was seen in the clinic not long ago whose condition clinically was a counterpart of that of Dr. Rulison's patient. He had rather prominent veins, over which he had no eruption. A case of similar involvement is mentioned in the German literature.

DR. R. H. RULISON: In regard to the matter of the intravenous injection of the dye, Nomland in his article stated that the selective affinity of congo red was developed by Bennhold as a test for generalized amyloidosis, but the reaction has been repeatedly negative when there was amyloidosis of the skin. In this case Dr. Greenhouse used the dye intravenously, and there was no staining of the cutaneous lesions. As to Dr. Fox's point, I purposely brought down several illustrations from the literature including those from Sutton's book (Diseases of the Skin, 1916 edition). I also have found the same illustration in all subsequent editions of the book. In the earlier edition the condition is called "lichen planus hypertrophicus, unusual type." In the later editions it is called lichen planus ocreaformis. At the end of an article Dr. Liebenthal uses the curious term "ocreaformis," which refers to the shin-guard distribution of the lesions. The pictures, which Sutton has produced in his book, to me look like amyloidosis cutis. I hope that the term "lichen planus ocreaformis" can be dropped from the literature. Apparently Liebenthal started its use.

As far as Dr. Oppenheim's question is concerned, the three conditions that seem to be confused in these cases are amyloidosis, some form of lichen planus and lichen simplex chronicus. One of the authors made a rather pertinent remark when he said that any long-continued condition of this type in which lichen planus of any form was suspected, if it were more marked or prominent on, or began on, the extensor surfaces rather than the flexor surfaces, should be considered as a possible amyloidosis cutis. The senile form of amyloidosis cutis is mentioned rather vaguely in the literature. This man is 72, but he has had his eruption for thirteen years and probably was not senile when it was first developed. Whether this is amyloidosis developing in an old lichen simplex chronicus I do not know.

DR. M. OPPENHEIM (by invitation): Amyloidosis circumscripta has been observed only in cases of lichen simplex chronicus and not in cases of lichen planus. Freudenthal, of Jadassohn's clinic, was the first to describe it. After his report came various reports from different places. It has been observed in cases of lichen simplex chronicus on the legs.

DR. R. H. RULISON: When this case was originally presented at the Bronx Dermatological Society, Dr. Satenstein, after examination of the slide, said that he saw no evidence of lichen planus, and lichen planus is one of the conditions that can be diagnosed from the microscopic picture.

Granuloma Annulare. Presented by DR. FRED WISE.

A. F., a girl aged 16, was presented on Jan. 24, 1939 (ARCH. DERMAT. & SYPH. **40**:164 [July] 1939), with granuloma annulare of the dorsum of the hand and disseminated pinhead-sized lesions on the extremities. At the time of presentation, I said that I did not know what histologic examination of these small lesions would show. The microscopic structure of these pinhead-sized lesions was characteristic of granuloma annulare. None of them has increased in size since the presentation two months ago.

Angioma Involving the Right Eye. Presented by DR. EUGENE F. TRAUB.

L. H., a woman aged 44, was presented on Nov. 22, 1938 (ARCH. DERMAT. & SYPH. **39**:763 [April] 1939), with a swelling involving the right eye. This lesion has completely disappeared, and the eye has returned to normal without any treatment. Previously she had had radium treatment before the disappearance of the lesion.

Erythema Figuratum Perstans. Presented by DR. FRANK C. COMBES.

C. C., a man aged 65, was presented on Jan. 24, 1939 (ARCH. DERMAT. & SYPH. **40**:167 [July] 1939), with circinate and gyrate lesions of twenty-five years' duration and untreated syphilis. Every one agreed with the diagnosis of erythema figuratum perstans, but one or two of the members suggested the possibility that the lesions had some relation to his syphilis in spite of the fact that there was no atrophy and no scaling.

A biopsy was performed.

DR. J. FRANK FRASER: I made a hasty examination of the slide but saw no features suggestive of syphilis.

DR. FRANK C. COMBES: The patient was very reluctant to take any treatment. He has been in good health all his life. He received two injections of a bismuth compound. After the first injection he became sick. He was finally persuaded to take the second one and again had a reaction. It did not affect the eruption. He refused oral treatment.

Epithelioma of the Upper Lip. Presented by DR. A. BENSON CANNON.

R. M., a woman aged 33, was presented on Feb. 28, 1939 (ARCH. DERMAT. & SYPH. **40**:510 [Sept.] 1939). The lesion on the upper lip proved to be a well differentiated carcinoma of low grade malignancy. The diagnosis was proved by microscopic examination by several pathologists.

J. FRANK FRASER, M.D., *Chairman*

April 25, 1939

FRANK C. COMBES, M.D., *Secretary*

Ichthyosis Hystrix in Two Brothers. Presented by DR. FRED WISE.

J. W., a boy aged 2 years, and his brother, K. W., aged 4 years, were registered at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital in September 1938. Since the previous presentation (ARCH. DERMAT. & SYPH. **36**:1286 [Dec.] 1937), the palms of both children had received a series of treatments with unfiltered roentgen rays, which resulted in only temporary removal of the severe hyperkeratosis.

Mycosis Fungoides. Presented by DR. FRED WISE.

Mrs. R. M., aged 70, applied for treatment on April 18, 1939. Sixteen years previously she had noticed an eruption on the trunk, which had consisted of disseminated nonelevated reddish brown plaques, was devoid of scaling, did not itch and gradually spread all over the body, exclusive of the face, palms and soles. According to her description, the lesions resembled those in parapsoriasis en plaques disséminées. In the past year painless elevated discoid plaques have appeared on the left forearm, the thighs and the legs.

Examination shows a discoid granuloma on the anterior aspect of the left forearm, 2 inches (5 cm.) in diameter. It is reddish brown, with the edge elevated and the central portion crusted. Within ½ inch (1.3 cm.) of this lesion are several pea-sized violaceous infiltrated papules. On the thighs and legs are scars of similar lesions which have been excised. The entire trunk and all four extremities show widespread brownish and coffee-colored discoid and oval plaques, varying from 1 to 5 inches (2.5 to 12.5 cm.) in diameter. They are slightly scaly, and one of them, on the right buttock, is slightly infiltrated. The patient complains of itching only when pointedly queried.

DISCUSSION

DR. EDWARD R. MALONEY: I believe that clinically this condition is typical of mycosis fungoides.

DR. JOSEPH J. ELLER: I agree with Dr. Maloney.

DR. FRANK C. COMBES: I agree with what Dr. Maloney has said with one exception, and that is that this patient says she has not had any itching. That

of course is unusual in a case of this type of mycosis fungoides in which there has been a prefungoid eruption for sixteen years. It is interesting, too, that this prefungoid eruption has many of the characteristics of parapsoriasis. I do not doubt that at some time a diagnosis of parapsoriasis was made.

DR. EUGENE F. TRAUB: The lesion on the forearm certainly looks like a typical fungating one. I think that the eruption on the body is more suggestive of poikiloderma vasculare atrophicans than of parapsoriasis. Poikiloderma vasculare atrophicans as the preceding eruption would account for the absence of itching.

A Case for Diagnosis (Pemphigus?). Presented by DR. EUGENE F. TRAUB.

E. S., a man aged 47, presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, is foreman in a varnish department, and while working in September 1938 he received a slight injury to the top of his head. The cut bled somewhat, and iodine was applied. The injury did not warrant his stopping work, nor did he consult a physician at the time. He treated the area, however, with ointments for about eight weeks. In October 1938 he consulted a physician, who assured him that the condition was not serious and gave him some ointment to apply. As the process did not disappear, he consulted a number of physicians, several of whom thought he might have pemphigus. He was given germanin intravenously, and either the medication or the illness caused him to feel sick and to have a fever. His appetite has been poor, and he has lost about 6 pounds (2.5 Kg.). He feels weak and has spells of cold sweating and indefinite pains in the legs. He has had hay fever for the past twenty-five years and also some asthmatic attacks. For the past eight years he has been receiving injections for these conditions annually, with resultant improvement. I present him for the purpose of obtaining a diagnosis and an opinion as to whether the accident that he described might have had any bearing on the present eruption.

DISCUSSION

DR. EUGENE F. TRAUB: This man is trying to claim compensation for his condition. He has been seen by several members of this society, and tentative diagnoses have been made. I do not want to do the man an injustice and say that there is absolutely no connection between his injury (the scratch he had on the head) and the eruption; but if it is pemphigus, as some of the members who have seen him think it might be, there probably is no connection with his work.

DR. EDWARD R. MALONEY: The case seemed to me to be one of either seborrhea or psoriasis. There were certain lesions which looked to me like lesions of psoriasis. I do not know on what the diagnosis of pemphigus was based. Certainly there are no vesicles present today, and if there were ever any, they must have been limited to that small area on the scalp. Were there any lesions at any time on the body?

DR. EUGENE F. TRAUB: The patient claims he had no lesions elsewhere at any time. When he was examined in the different clinics and offices, the skin of his back was pinched, probably in looking for a Nikolsky sign, and the patient claims that lesions arose on the sites of these pinched areas. He had only an eruption on the scalp on the two occasions I have seen him, with the possible exception of two very doubtful vesicles in the groin.

DR. EDWARD R. MALONEY: The crusting appears to me to be the crusting of seborrhea.

DR. PAUL E. BECHET: The patient presents a scaly lesion on the top of the scalp. It is not infrequently that one sees a secondary infection in addition to a seborrheic eczema, which spreads at times to the axillary and genital regions. I should be inclined to call this case one of seborrheic eczema with secondary infection.

DR. FRED WISE: I saw this man some time ago, and at that time the lesions were vesicular and bullous, and my impression was that he had pemphigus. I still think he has pemphigus.

DR. A. BENSON CANNON: I am of the opinion that the palm-sized area of erythema and crusting on the front of the patient's scalp has been produced with an idea of obtaining compensation. While I believe that Dr. Wise is right in suspecting a beginning pemphigus, especially as it often has its origin in the scalp, I believe that such a diagnosis is remote. It is difficult for me to believe that pemphigus would remain confined to one small area for so long a time.

DR. R. H. RULISON: The eruption on the scalp suggests a seborrheic lesion that has been scratched. I cannot get rid of the thought that the eruption is self-perpetuated.

DR. FRANK C. COMBES: I am probably prejudiced by the fact that this is a compensation case, but I agree entirely with what Dr. Cannon has said. I think that this condition is seborrheic eczema that has been traumatized and that there is a secondary staphylococcic infection. The patient picks at his scalp. The other lesions are bizarre, such as the little crusts to which he drew our attention on his back. They are peculiar and not suggestive of any dermatosis. He also draws attention to a mild dermatitis on the heel, which I do not think has any connection with the other lesions.

DR. EUGENE F. TRAUB: I do not believe this man is a malingerer. He has not seemed so to me on the two occasions on which I have seen him. He is certainly impressed with the diagnosis of pemphigus. After the injections of germanin, which evidently made him ill, his condition was much worse, and I do not know whether the improvement that followed was the result of the germanin or whether it was just one of those remissions that occur spontaneously. However, I cannot make a diagnosis of pemphigus on the basis of the appearance of the eruption today.

Psoriasis of the Face and Palms. Presented by DR. EUGENE F. TRAUB.

C. P., a woman aged 27, from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, presents a bandlike eruption about the mouth and an extensive eruption on both hands, involving particularly the entire palmar surfaces. The eruption has been present about the mouth between five and six years and has more recently appeared on the hands after a sunburn. There are also a few lesions on the scalp and elsewhere on the body. The patient has had between sixteen and seventeen treatments with roentgen rays on the face, without relief. She has also had innumerable treatments with ultraviolet radiation, injections of preparations of bismuth and arsenic and one injection of a gold compound. All the various local remedies have proved ineffective. I have given the patient intravenous injections of sodium thiosulfate and roentgen treatments of the hands, together with a soothing ointment. There was slight improvement to begin with, and then the process reached a stationary point. The patient is presented for the purpose of obtaining suggestions for treatment.

DISCUSSION

DR. R. H. RULISON: I do not know of any therapy which will help this condition.

DR. A. BENSON CANNON: While I agree with Dr. Rulison that palmar and plantar psoriasis is most resistant to all forms of treatment, I have seen such lesions disappear under the influence of sunlight. I should advocate sending the patient to a warm climate where she would have the benefit of sun baths at the beach for a number of months, so that her skin would become thoroughly tanned. Of course, it is also necessary for the patient to be examined to make certain that she has no physical condition which would tend to aggravate the psoriasis,

such as a focus of infection, secondary anemia or excessive arsenic in the system. I believe that Drs. Kingsbury and Myers found large quantities of arsenic in the blood and urine of persons suffering from psoriasis of the palms and soles and reported good results after the use of sodium thiosulfate. While I have had no experience with sodium thiosulfate in the treatment of such a condition, it might be worth while to investigate the possibility of arsenic in this case.

DR. EDWARD R. MALONEY: The only suggestion I have to make is that Dr. Traub might possibly try a diet high in vitamins, particularly vitamin C. Some reports have been made that psoriasis has cleared up with the use of vitamin C, or ascorbic acid.

DR. JOSEPH J. ELLER: I think, as Dr. Cannon does, that general improvement in the health is the only way to clear up a condition of this type. The patient would certainly benefit by six months or a year of outdoor life in a place such as Florida, if that were possible. A high caloric, high vitamin diet, sunshine and so forth sometimes make a recalcitrant condition amenable to treatment.

DR. EUGENE F. TRAUB: The patient has received some sodium thiosulfate, with the idea that the arsenic she had in the beginning might have some bearing on the eruption. However, there has been no improvement so far. I will try the high vitamin diet. She is a poor working girl, and I doubt if she could go to Florida.

A Case for Diagnosis (Nodular Neurodermatitis?). Presented by DR. FRED WISE.

S. M., a woman aged 24, was previously presented by Dr. Marion B. Sulzberger at the Bronx Dermatological Society on April 28, 1938 (ARCH. DERMAT. & SYPH. **38**:666 [Oct.] 1938). Since the prior presentation, all signs of papillomatosis have vanished. At present the lesions are pruritic, hyperpigmented, elevated, nodular, with lichenification of their surfaces, and from 5 to 20 mm. in diameter. They are on the abdomen, chest and thighs. There is an occasional attack of urticaria, but not at the present time.

DISCUSSION

DR. JOSEPH J. ELLER: The lesions remind me of urticaria pigmentosa acquisita. The girl says that during attacks of urticaria the lesions sometimes swell.

DR. PAUL E. BECHET: I think that this case is the most interesting one presented today. I have no diagnosis to offer. My experience with acanthosis nigricans is limited. I have seen only a few adults with the disease, and in all of them carcinoma of the internal organs was demonstrated. Despite this I believe that acanthosis nigricans does not necessarily have to be associated with malignancy, and it seems to me that Dr. Wise's case is one of acanthosis nigricans.

DR. J. GARDNER HOPKINS: Can this condition be a lichenoid neurodermatitis?

DR. EUGENE F. TRAUB: If the condition is not a lichenoid neurodermatitis, it might be some peculiar abortive type of juvenile acanthosis nigricans. I have a patient now who has an eruption resembling this one, and the histologic diagnosis is definitely acanthosis nigricans. We do not observe such cases often, and I imagine that there are some variations in the picture.

DR. FRED WISE: It is no doubt evident that the patient had two distinct changes in the skin. She had at one time crusted lesions on the scalp which resembled lesions of a long-standing untreated favus. Such a condition certainly is not typical of urticaria pigmentosa. In the histologic sections there were no abnormal amounts of mast cells. Some time ago she had lesions under the breasts and in the axillas which aroused the suspicion of acanthosis nigricans, but they have now disappeared. The only diagnosis I have to offer which agrees with the histologic picture is nodular neurodermatitis, as described by Pautrier.

Arsenical Keratoses. Presented by Dr. Paul E. Bechet.

G. D., a man aged 25, came to the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on April 18, 1939, with a generalized eruption of two years' duration. For several years before the appearance of the eruption and for months at a time he had been taking about 30 drops of solution of potassium arsenite U. S. P. (Fowler's solution) a day, in order to relieve a chronic acne.

The patient presents a severe hyperkeratotic thickening of the skin of the palms and soles. On the dorsal aspect of the fingers and hands are innumerable wart-like protuberances. There are pigmentary changes on the trunk, and the hair is dry, brittle and sparse. The entire cutaneous surface is dry. The eyelids are granular and puffy.

The Wassermann and Kahn reactions were negative. A blood count was about normal. There has not yet been enough time for reports to be completed on the examinations of the twenty-four hour specimen of urine for arsenic and of the histologic sections of skin.

DISCUSSION

Dr. Paul E. Bechet: The interesting feature of this case is the therapy. The patient is young and so is the arsenical keratosis. It is severe, and it will undoubtedly increase with the years. Therefore, something must be done. While I have had somewhat disappointing results with sodium thiosulfate in the treatment of acute toxic arsenical eruptions, such as the arsphenamine dermatitides, the results have been excellent in the treatment of chronic arsenical intoxication. In several instances the long-continued administration of sodium thiosulfate, both by the intravenous and by the oral route, has succeeded in diminishing the arsenical content in the urine and in greatly ameliorating the eruption. The keratoses as well as the pigmentary changes have improved, and the skin has become smoother and less dry. I believe that in order to achieve such good results, one must use the drug over a prolonged period. Even after months of administration of sodium thiosulfate in large doses, I have never seen any deleterious effects.

A Case for Diagnosis (Lupus Miliaris Disseminatus Faciei?). Presented by Dr. Eugene F. Traub.

M. F., a woman aged 24, came to the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital in March 1939, complaining of an eruption about the nose and mouth of six years' duration. The eruption consisted of tiny dots of pigmentation which on close inspection proved to be small papules which healed leaving tiny pitted scars. A larger lesion on the chin, which somewhat suggested the appearance of a common mole, resulted, according to the patient's statement, because she had squeezed what appeared to be a tiny "pimple." No local therapy seemed to have any effect on the condition, and the usual remedies applied for treatment of seborrhea actually seemed to aggravate the eruption. Tuberculin tests with dilutions of 1 to 1,000,000, 1 to 100,000 and 1 to 10,000 all gave 1 plus reactions. The larger papule on the chin, after removal and sectioning, was reported by Dr. Barthel as a chronic inflammatory nodule and by Dr. Satenstein as an intensive inflammatory type of lupus vulgaris.

DISCUSSION

Dr. Fred Wise: If I were to make a diagnosis from the section, I should consider an unusual form of disseminated miliary tuberculosis. The clinical interpretation of this eruption is difficult.

Dr. J. Gardner Hopkins: All that I could observe in the histologic examination was a picture of chronic tuberculoid granuloma, which can also be observed in chronic acne. There were no very good tubercles. I noticed a foreign body in one giant cell. There was a tremendous lymphocytic infiltration.

DR. J. FRANK FRASER: The presence of epithelioid and giant cells suggests tuberculosis, but I should not make a positive diagnosis, either from the clinical or from the histologic picture, without further study. I should advise tuberculin tests and inoculation of animals. There are various granulomas of the skin which may resemble tuberculosis.

DR. FRED WISE: I should not make that diagnosis clinically, without the aid of the histologic picture.

DR. EDWARD R. MALONEY: I cannot see anything in this case from the clinical standpoint that justifies a diagnosis of tuberculosis or of tuberculid. If either of those diagnoses is made, it must be made histologically.

DR. JOSEPH J. ELLER: I agree with Dr. Maloney.

DR. JEROME KINGSBURY: I think this condition is benign. I believe that clinically it is follicular seborrhea.

DR. R. H. RULISON: I agree with Dr. Kingsbury. It seems to me that while the symptoms are a little unusual, they could be explained on the basis of a seborrheic condition. The patient has not had any roentgen therapy, has she?

DR. EUGENE F. TRAUB: Clinically the condition was considered as possibly a peculiar type of perioral pigmentation of the type that Brock described, but that diagnosis did not seem correct because of the peculiar little papules. They are definitely papules. I have seen this patient at least six or eight times and have never seen any pustules. There are a peculiar diffuse pigmentation or browning of the skin and a little redness, and in the daylight the old lesions that have healed show as tiny atrophic spots. Therefore, the lesions apparently heal with tiny pitted scars. Adenoma sebaceum was also thought of, except that in this case the eruption is more extensive on the chin than one usually sees and less extensive, comparatively, around the nose. The condition in this patient has been present for six years, and has been subjected to all sorts of treatment for seborrhea, including application of salicylic acid and sulfur, which made it worse.

When the specimen was sent to the laboratory, I wanted to exclude, for one thing, some type of cutaneous tuberculosis. The one large lesion looked very much like a mole, and clinically one could not be sure it was not a mole. The lesion removed for microscopic examination was much larger and different from the others that are present. It was removed with an elliptic excision, and a suture was in, because I thought this method would give a better cosmetic result than removal with a punch. The excision was done only two weeks ago, and sufficient time has not elapsed to see if the lesion will recur, as it might do in lupus vulgaris.

Report on a Case of Psoriasis Amiantacea. Presented by DR. FRED WISE.

J. C., a man aged 60, was presented before the Society on March 28, 1939 (ARCH. DERMAT. & SYPH., this issue, p. 1031), with the diagnosis of psoriasis amiantacea. Dr. Oppenheim suggested at the previous meeting the diagnosis of Weidenfeld's dermatitis verrucosa. Dr. Satenstein has examined the histologic section and pronounced the condition to be typical psoriasis.

Report on a Case for Diagnosis (Duhring's Disease?). Presented by DR. FRED WISE.

E. M., a woman aged 49, was presented before the Society on March 28, 1939 (ARCH. DERMAT. & SYPH., this issue, p. 1030), with the tentative diagnosis of Duhring's disease. The diagnosis made by Dr. Satenstein from study of the histologic section was Duhring's disease, as far as he could tell. No other diagnosis was considered tenable.

Report on a Case of Atrophia Cutis Maculosa. Presented by DR. FRED WISE.

A. M., a woman aged 48, was presented before this society on March 28, 1939 (ARCH. DERMAT. & SYPH., this issue, p. 1029) as having atrophia cutis maculosa. After examination of the histologic section, Dr. Satenstein reported that the condition was apparently a primary atrophy of the skin.

Report on a Case for Diagnosis (Sporotrichosis?). Presented by DR. EDWARD R. MALONEY.

C. de S., a man aged 48, was presented before the Society on March 28, 1939 (ARCH. DERMAT. & SYPH., this issue, p. 1033) with the tentative diagnosis of sporotrichosis. The diagnosis has not yet been proved. No cultures have given positive results. There has been no evidence of sporotrichosis in the animals which were inoculated. It is about time to kill some of them for examination.

Report on a Case of Epithelioma of the Upper Lip. Presented by DR. A. BENSON CANNON.

R. M., a woman aged 32, was previously presented on Feb. 28, 1939 (ARCH. DERMAT. & SYPH. **40**:510 [Sept.] 1939), and the histologic observations were reported on March 28, 1939. The patient was admitted to the Presbyterian Hospital, where she was observed by Dr. Stevenson and several other surgeons in consultation. They all agreed that the lesion was clinically and histologically a squamous cell epithelioma and that probably a secondary infection accounted for its rapid growth. Excision of the growth was not thought advisable, because of the resulting deformity of the upper lip. Therefore, daily fractional doses of roentgen rays were given. The lesion has almost disappeared during the last month, and it is my opinion that there is a probability of a permanent cure.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILOLOGY

EUGENE F. TRAUB, M.D., *Chairman*

April 4, 1939

LOUIS TULIPAN, M.D., *Secretary*

Neurogenic Sarcoma of the Foot. Presented by DR. ANDREW H. MONTGOMERY.

P. L., a Negress aged 51, is presented from the New York Polyclinic Hospital, where she was first seen a few weeks ago. She has had a growth on the dorsum of the right foot for the past fourteen years. Originally it was a small nodule. Four years ago it was traumatized and began to grow. At the time of the first examination, there was a flattened, rounded, silver-dollar–sized, grayish brown, mushroom-like tumor of firm consistency in front of the right external malleolus. The lesion was slightly pedunculated, the lower half having a rolled border. The surface was broken by a large vertical ulcer, from the center of which a deep fissure extended to the anterior border. There was a purulent discharge with a foul odor. The growth was partly fixed to the underlying structures, especially to the third extensor tendon. A biopsy specimen, including the ulcer, was taken for examination. Under wet dressings the inflammation has subsided, and the lesion has shrunken appreciably and is more freely movable. Subjective symptoms have been negligible.

Microscopic examination of the specimen removed showed a tumor of the skin, with some keratinization of the surface epithelium and with a tendency toward ulceration. There was some hemorrhage on the surface. Beneath it was a tumor of neurogenic origin which was poorly circumscribed. The fibers were curled and interlocked in a fashion common to neurogenic tissue, with a tendency toward the production of small whorls and ill defined fasciculi. There was decided vacuolation as the result of the lipoid present, and the general character was that of neurogenic tissue of sheath of Schwann type. The fat was equivalent to myelin. As a whole, it was fairly well differentiated but was believed to possess low grade malignant possibilities of borderline type. The histologic diagnosis was neurogenic sarcoma of the skin.

The Wassermann reaction with cholesterolized and alcoholic antigens was negative. The Kahn reaction was also negative.

DISCUSSION

DR. ANDREW H. MONTGOMERY: This case is one of the few in which all the histologists who have examined the lesion have agreed on the diagnosis. There

Fig. 1.—Neurogenic sarcoma of the foot.

has been a certain amount of responsibility in keeping the patient for this presentation. The lesion will probably be excised.

DR. CHARLES WOLF: In contrast to the condition in this case, neurogenic sarcoma, especially of the bones, is a malignant process. In the few cases I have observed, a rapid extension has been the usual course. It seems that this particular lesion is a slowly growing one, and perhaps excision will result in a cure. However, I should also ascertain whether there are any metastases at the present time, especially in the lung. I should also follow the excision with massive doses of filtered roentgen rays locally, about 6,000 r at high voltage.

Lepra (Neural Type). Presented by DR. ANDREW H. MONTGOMERY.

K. K., an Armenian aged 47, who has been in the United States for thirty years, is presented from the New York Polyclinic Hospital. He began to "feel sick" about seven years ago and has been treated in various clinics. The patient has a leonine expression and has many infiltrated papules scattered over the face and ears. On the left hand the outer three fingers are atrophic. The skin is shiny and anesthetic. Neurologic examination showed the ulnar nerves to be

enlarged, especially on the right side, with varying degrees of anesthesia involving the upper part of the face, arms, forearms, trunk, thighs and legs.

Examination of tissue from a nodule on the posterior surface of the lobe of the right ear showed the presence of tubercles and vacuolation of cells about the vessels. Acid-fast stain on this tissue failed to show the lepra bacillus. Smears of material from the nose and throat also failed to reveal this bacillus.

DISCUSSION

DR. ANDREW H. MONTGOMERY: There are practically no cutaneous lesions. According to the pathologists' general report on the nodule excised from behind the ear, nothing significant was observed. Dr. Satenstein said that he would definitely make the diagnosis of leprosy from the histologic section. I think that on the basis of the cutaneous anesthesia alone one can make the diagnosis of leprosy.

DR. EUGENE T. BERNSTEIN: I do not think that this case is one of syringomyelia, because the patient does not show a Brown-Séquard syndrome. The interosseous spaces are not affected. I should not consider this case one of leprosy without observing the lepra bacillus.

DR. ANDREW H. MONTGOMERY: This condition is almost a purely neural type of leprosy. The cutaneous lesions are negligible. Opposing a diagnosis of syringomyelia is the fact that this condition is usually limited to the arms and the upper levels of the trunk. The trouble is usually in the cervical portion of the spinal cord. This man has atrophy of the small muscles of the left hand, with beginning contraction. I do not know what other condition than leprosy would give him the widely distributed anesthesia.

A Case for Diagnosis (Lymphangiectasis?). Presented by DR. ANDREW H. MONTGOMERY.

J. F., a woman aged 37, is presented from the New York Polyclinic Hospital because of a condition involving the right deltoid region, extending down the right arm to the elbow, of two years' duration. There is a diffuse mottled eruption on the outer side of the right arm in the deltoid area and just below, consisting of slightly elevated and rounded, pouchlike soft flesh-colored papules, pea sized or larger. They are more prominent in the dependent position.

Roentgenographic examination in August 1938 showed no deviation from the normal in the bones or joints of the cervical part of the spine. There was no evidence of cervical ribs. Examination of the shoulders, clavicles and arms showed a large irregular area of calcification in the soft tissues overlying the tuberosity of the humerus on the right side, consistent with extensive calcification of the subacromial bursa. The conclusion was that there was extensive calcification of the right subacromial bursa. Reexamination on March 24, 1939, showed a large periarticular calcification in the region of the right deltoid bursa. This calcification was triangular, and its greater diameter was approximately ¾ inch (2 cm.). There was a smaller plaquelike area of calcification just external to this larger calcified area. Some decalcification of the head of the humerus was noted in the deltoid area, which was associated with the bursitis. As this type of bursitis may cause general changes in the soft tissue about the joint, the observations noted may be the underlying pathologic cause of the superficial lymphatic stasis.

Microscopic examination showed a mild scaly desquamative lesion, with slight thinning and atrophy of the epidermis and slight pigmentation of the basal layer. A few of the superficial blood vessels were surrounded by a slightly edematous structure containing a few lymphocytes. In reality, however, the lesion showed very little. At one point there seemed to be a slight proliferation of the small nerve bundles in the midportion of the corium. It could easily be called scar tissue, but the pathologist was inclined to believe that the most outstanding feature

of the section was the proliferation of the nerve elements and that the lesion was not primarily inflammatory or lymphatic. The condition belonged in the category of proliferative disturbances, in the nervous system, different from but related to von Recklinghausen's disease. The diagnosis was subcuticular neurofibromatosis.

DISCUSSION

DR. FRED WISE: This patient had previously attended the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, where the histologic diagnosis made by Dr. Satenstein, without his knowing the clinical features, was fibroneuroma or possibly von Recklinghausen's disease. There were no signs of ectasia of the lymphatic vessels. Dr. Montgomery's suggestion of the presence of some obstruction to the lymph circulation is to be taken into consideration, because the patient says the lesions are spreading. Therefore, I think further study must be made. The diagnosis of von Recklinghausen's tumor can be made only with special stains of the nerve fibers. In this instance such special stains should be used to see if there is any infiltration of the neurilemma.

DR. ANDREW H. MONTGOMERY: I do not know what the diagnosis is in this case. I feel that this condition is due to some lymphatic obstruction in the shoulder joint. The patient has had a subacromial bursitis. I am reasonably sure that the shoulder joint is the site of the trouble.

A Case for Diagnosis (Urethral Ulcer). Presented by DR. FRANK VERO.

G. M., a man aged 38, was first seen in the department of urology of the New York Polyclinic Hospital in October 1938, with complaints of difficulty in urination and ulceration and swelling of the urethral meatus of three or four months' duration. He said he had not had any previous venereal infection but that the present condition followed an extramarital intercourse. He resisted all local and systemic medication and was referred to the department of dermatology about one month ago.

Examination showed a depressed, atrophic scar at the meatus and a wide gaping of the urethra, exposing an extensive ulceration, especially at the floor. The ulceration was covered with a serofibrinous yellowish membrane. On palpation the surrounding tissues, extending about ¾ inch (2 cm.) from the meatus, were indurated and slightly tender. The inguinal glands were slightly enlarged, hard and nontender. For the past four weeks the patient has received only sulfanilamide by mouth and local baths in solution of potassium permanganate, with only slight improvement in his condition.

Repeated Wassermann and Kahn tests gave negative reactions. Dark field examination gave negative results. Examination revealed no Ducrey bacilli or gonococci. A smear for Donovan bodies gave negative results. Histologic examination of a specimen from the urinary meatus with a small ulceration with rolled white edges showed a portion of mucous membrane or skin presenting inflammatory ulceration with a well marked inflammatory reaction which extended underneath the margins of the normal mucosa. There were no signs of a neoplasm. There was an occasional small multinucleated giant cell, and one focus showed a moderate degree of perivascular lymphocytic infiltration. The giant cells were apparently of the foreign body type, and the picture did not suggest tuberculosis. The histologic diagnosis was chronic granulomatous ulcer of the penis.

DISCUSSION

DR. HERMAN GOODMAN: I think this case is one of malingering. This man's makeup, his tremendous interest in himself and his exhibitionism should be taken into consideration. He brought a flashlight to help display the lesion. He gives his story of the lesion. All this points to the diagnosis of a factitious lesion. The ingenious device Dr. Lapowski recommended should be used: a test tube to cover the penis and protect it from trauma.

DR. CHARLES WOLF: I believe that there is the possibility of an artefact in this case.

DR. E. W. ABRAMOWITZ: I agree that there is a factitious element present, but I think that a possible epithelioma or other serious condition should be watched for.

DR. FRED WISE: I should bear in mind the possibility of tuberculosis orificialis developing, because the urethral meatus is a favorite location for it. The mere fact that the biopsy did not show tubercle bacilli is not enough to exclude that diagnosis.

DR. EUGENE F. TRAUB: I also think that the lesion is possibly an artefact, although it is a condition which should be watched.

DR. FRANK VERO: Of course this case needs further study. The possibility of tuberculosis has also been considered in the clinic and will be investigated. I agree with Dr. Goodman that there may be a factitious element present. This patient likes to exhibit his genitals.

NOTE.—Since the presentation of this case, a Frei test and a Dmelcos test have been performed, giving negative reactions.

Perifolliculitis Abscedens et Suffodiens, with Secondary Cutis Verticis Gyrata. Presented by DR. HENRY D. NILES.

F. M., a man aged 32, born in the United States, is presented from the New York Hospital, with a condition of the scalp which has been present for fourteen years but has been worse for the past seven years. The patient states that his scalp was normal to the age of 18. The eruption began with itching of the scalp, without any lesions or dandruff. Since then he has had many small and large deep follicular pustules and severe exudation of the scalp from time to time. Healing of the pustules has been followed by irregular areas of scar tissue, alopecia and furrows. On two occasions he has had a generalized neurodermatitis and involvement of the nails, especially that of the right ring finger. The patient states that his hair began to fall out after one roentgen ray treatment about two years ago.

The patient is a moron with hypogonadism. The scalp is uneven and furrowed and covered with many grooves and ridges extending from the center to the back of the scalp. There are several patches of partial alopecia containing short hairs, some small follicular pustules and large boggy and indurated swellings. The anterior part of the scalp is clear, but there is a large swelling over the occiput. There are no definite areas of scar tissue. Longitudinal ridges and furrows of thickened scalp resemble cutis verticis gyrata. At the present time the scalp is in a relatively good condition, and there are no large abscesses and no exudation.

The results of urinalysis and Wassermann tests were negative. A blood count was normal. Histologic examination of a section removed from the scalp on Dec. 8, 1938, showed moderate lymphocytic infiltration in the corium, mostly around the follicles, and moderate hypertrophy of the sebaceous glands but no acute inflammation, suppuration, giant cells or granulomatous reaction. In some areas there was a perivascular infiltrate. The picture was that of a nonspecific chronic inflammation of the corium, mainly a perifolliculitis but not that of perifolliculitis capitis abscedens et suffodiens. Roentgenograms of the forearms showed a bony fusion of the upper ends of the radius and ulna.

DISCUSSION

DR. EUGENE T. BERNSTEIN: It impressed me first that this was a case of cutis verticis gyrata, associated with follicular lesions, which are usually seen in patients with neurodermatitis. I do not think there are enough active lesions or large lesions to justify a diagnosis of perifolliculitis capitis abscedens et suffodiens.

DR. E. W. ABRAMOWITZ: Perifolliculitis capitis abscedens et suffodiens is obstinate to treatment. One may still observe considerable activity, with undermining, even after years of treatment. As there are no active lesions present, this condition may be an acquired form of cutis verticis gyrata resulting from a long-continued inflammation of the scalp. An underlying true (nevoid) cutis verticis gyrata with secondary infection should be considered also.

DR. R. H. RULISON: I agree with Dr. Abramowitz.

DR. LOUIS TULIPAN: I believe that the diagnosis is perifolliculitis capitis abscedens et suffodiens. In the first place, the patient has the typical boggy masses. Secondly, he is losing hair and there is scarring, which is not observed in cutis verticis gyrata. In the latter the skin of the scalp is absolutely normal except for the folds which give the appearance of folds of skin, almost like the convolutions of the brain. This man has boggy masses without the folds in the skin, and as I said, the skin is affected, showing inflammation and scars. It looks as though there had been abscesses there previously. I do not believe that cutis verticis gyrata would affect the entire scalp. Of course, it is seen sometimes, but it is usually only on the posterior part of the scalp, like the folds of the scalp hanging down over the neck of a fat person.

DR. FRED WISE: This patient does not have any folds of the scalp. He has one ridge extending from the occiput to the vertex. There are no gyrate formations. In cutis verticis gyrata the folds may be in any direction, vertical, transverse or oblique. I have not made up my mind about the diagnosis in this case because I think a better history, as of a preceding eruption, is necessary in order to come to any conclusion.

DR. TIMOTHY J. RIORDAN: It seems to me that trichotillomania should be considered because of the loss of hair. Part of the hair on one side looks as though it had been either pulled out or rubbed out.

DR. EUGENE F. TRAUB: I too was interested in that point. Some of the hair has been cut in the process of treatment, or possibly a little thinning may have occurred as a result of the roentgen ray treatment, although this is doubtful. Some of the hair has undoubtedly been actually worn away by constant scratching. Fisher stated the belief that this anomaly is developmental in character and is comparable to the condition present in the heads of many mammals, such as lions, tigers and bulldogs. He stated the belief that this type constitutes the only true cutis verticis gyrata. In another group he designates those conditions of the scalp which appear in the form of cutis verticis gyrata. These comprise conditions due to (1) inflammatory changes induced usually by micro-organisms; (2) anomalous and apparently congenital hypertrophies of the connective tissue; (3) tumors, such as fibromas, neurofibromas and cellular nevi, and (4) changes in the scalp occasioned by diseases that lead to proliferation of the tissues, examples of which are acromegaly, leukemia, myxedema and syphilis. In view of this classification and the fact that the patient presented certainly does not have any suppurating abscesses, it seems to me that his condition should be classified in the group which Fisher really designated as pseudo cutis verticis gyrata. The fact that it apparently began with an inflammatory or infectious process would in no way exclude such a diagnosis.

DR. HENRY D. NILES: Of course, the history from the patient would be unreliable, but his mother comes to the clinic with him. She is an intelligent person, and she is absolutely certain that the boy's scalp was normal until he was about 14 years of age, with no furrows or irregularity. After that age, the condition began with some redness and pustules and oozing. The longer the inflammatory process continued, the more furrows and ridges and irregularities developed, which would support Dr. Traub's contention. I admit that the scalp is certainly not typical tonight. My conception of this case is that there is a distortion of the scalp which is secondary to the inflammatory condition which he had.

Multiple Sarcoids. Presented by Dr. Henry D. Niles.

I. W., a Negro aged 39, born in the British West Indies, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital. The eruption is of three years' duration and involves the mouth, the back of the neck, the right ring finger, the perineum, the right axilla and the back. On the nape of the neck is a palm-sized, circumscribed lesion, with a raised infiltrated border and a red, somewhat atrophic center. A similar lesion extends from the nose around the mouth and to the chin, and another is in the posterior part of the right axilla and in the perineum. There are two or three dime-sized patches on the upper part of the back. There are no lesions on the genitalia or in the mouth. There is generalized glandular enlargement, especially of the femoral and inguinal glands. There is a large soft cystic swelling of the soft tissue on the dorsum of the right ring finger.

The patient says he has not had any venereal disease. He is in good general health. Because of a 2 plus Wassermann reaction the patient has had intermittent antisyphilitic treatment for the past three years, with little, if any, improvement in the cutaneous lesions. His wife and 2 children have negative Wassermann reactions, and his wife has had no miscarriages.

The Wassermann reaction was negative. The Kahn reaction was plus-minus. Reaction to tuberculin tests with dilutions of 1 to 1,000,000, 1 to 100,000 and 1 to 10,000 were negative; with a dilution of 1 to 5,000, 1 plus; with a dilution of 1 to 1,000, 2 plus, and with a dilution of 1 to 500, 2 to 3 plus. A blood count showed 79 per cent hemoglobin, 4,400,000 red blood cells per cubic millimeter and 6,800 white blood cells per cubic millimeter, with a differential count of 66 per cent polymorphonuclear leukocytes, 26 per cent lymphocytes and 4 per cent eosinophils. A roentgenogram of the chest was reported on as follows: "The picture is devoid of definite etiologic characteristics." There was slight enlargement of the heart and of the aortic arch. A roentgenogram of the right ring finger showed cystic-like degenerative changes, and moderate expansion was discernible, especially at the base of the proximal phalanx. Histologic examination of a section taken from the lesion on the neck showed a typical picture of sarcoid.

DISCUSSION

Dr. Eugene T. Bernstein: There are many features in favor of and many against the diagnosis of sarcoidosis. In the first place, there are many broken-down, ulcer-like lesions which may suggest tuberculosis cutis, like lupus vulgaris. Then, there are some lesions on the mouth which I should consider as tuberculosis psoriasiformis. There is also an involvement of only the one finger with so-called tuberculosis cystica. This condition is usually a multiple involvement in tuberculous osteitis multiplex cystica. In favor of a diagnosis of sarcoidosis is first, of course, the histologic picture. Is this a case of lupus erythematosus? That is sometimes considered generalized sarcoidosis. The patient also has lesions around the anus which I should consider to be tuberculosis cutis. It is difficult to draw a distinction between the picture of tuberculosis cutis and that of syphilis. Syphilis should always be under consideration. My final diagnosis in this case is tuberculosis cutis with a question mark. Concerning the Mantoux test, the results with dilutions of 100,000 and 1,000,000 were negative, which of course would favor the diagnosis of sarcoidosis, because the condition here is a positive anergy.

Dr. Henry D. Niles: There is one aspect which was not discussed, namely, the generalized adenopathy, which seemed to me very unusual in this type of case. I do not remember having seen it before. As Dr. Bernstein said, I think that this is an example of the many manifestations which a tuberculous infection can take in the Negro. The condition has been mentioned particularly recently by Dr. Bloom. The patient has an eruption resembling lupus erythematosus on the face, lesions similar to tuberculosis orificialis around the anus and marked general enlargement of the lymph glands. It is also interesting that he showed

no response to arsenical therapy, which is an advocated treatment for sarcoid. At present he has a negative Wassermann reaction and a plus-minus Kahn reaction, but this is after three years of treatment. He has not had an examination of the spinal fluid as yet.

Tuberculosis Cutis (Lupus Miliaris Disseminatus). Presented by DR. TIMOTHY J. RIORDAN.

S. F., a Spanish South American woman aged 38, is presented from St. Vincent's Hospital. She was first seen in the outpatient department, on Aug. 25, 1937. The Kahn and Wassermann reactions were 4 plus, and antisyphilitic therapy was started for latent late syphilis. On August 1938 she presented a generalized eruption consisting of nonpruritic pea-sized papules. Some of the lesions were shiny and flat, while others were topped by a hemorrhagic crust. She said that she was taking a medicine for nervousness, a liquid called "Bromadura."

A blood count showed 60 per cent hemoglobin, 3,980,000 red blood cells per cubic millimeter and 7,350 white blood cells per cubic millimeter, with a differential count of 67 per cent polymorphonuclear leukocytes, 1 per cent metamyelocytes I and 5 per cent metamyelocytes II, 25 per cent lymphocytes, 1 per cent eosinophils and 1 per cent monocytes. The red blood cells showed a moderate degree of achromasia and anisocytosis. Urinalysis was normal. No examination was made for bromides. A roentgenogram of the chest showed considerable congestion in both lungs, extending from the root areas into the midperipheral zone and the base; the heart measured 13 cm., the normal for this patient being 12 cm. There were no abnormal changes in density or contour of the bones in the roentgenograms. The results of tuberculin tests with dilutions of 1 to 1,000,000 and 1 to 100,000 were negative, and with a dilution of 1 to 1,000, positive. Histologic examination showed productive tuberculosis. In the corium there was a large conglomerate tubercle consisting of giant cells and epithelioid cells. The overlying epidermis was thin and about to rupture.

DISCUSSION

DR. FRED WISE: The condition in this case is difficult to diagnose from a morphologic point of view. The histologic study shows a tuberculous structure, but the lesions on the forearm certainly resemble a papulonecrotic tuberculid; and it is unusual to encounter the histologic picture of true tubercle formation in a papulonecrotic tuberculid. However, similar instances have been described, so there is a possibility of a coincident papulonecrotic tuberculid and disseminated miliary lupus. It would be interesting to obtain biopsy specimens from other lesions to see if the same tuberculous structure exists in them. I accept the diagnosis as presented.

DR. DAVID BLOOM: Of interest to me is the fact that this tuberculous eruption appeared during antisyphilitic therapy. I have presented before this Society (ARCH. DERMAT. & SYPH. **25**:566 [March] 1932) a similar case, in which an eruption, diagnosed by microscopic examination as lupus miliaris disseminatus, developed in a white woman while she was under treatment for secondary syphilis. In many Negro patients cutaneous tuberculosis has appeared while the patient was receiving antisyphilitic therapy. One is impressed with the thought that antisyphilitic remedies may be a factor in provoking the cutaneous manifestations of tuberculosis in these patients.

A Case for Diagnosis (Epithelioma Morphoeicum?). Presented by DR. HERMAN GOODMAN.

D. L., a woman aged 37, mother of 3 children, is presented for diagnosis. A cutaneous infiltration is observed involving the right ala nasi fold contiguous with the cheek. It has been present for about two and one-half years without

change other than a slight increase in diameter. The lesion is single and lies in the skin close to the ala nasi. There is a suggestion that the periphery is raised and the center depressed. Its diameter is about three-eighths inch (1 cm.). The color is mildly yellowish. The vessels over the lesion are telangiectatic. There is no visible evidence of a pearly border. Treatment has consisted of local greasy applications.

The patient is presented for clinical diagnosis as she is reluctant to have a biopsy made unless it is absolutely necessary. Clinical diagnoses considered have been localized scleroderma with telangiectasis and sclerodermatous-like epithelioma.

DISCUSSION

DR. CHARLES WOLF: The lesion is shiny, showing telangiectasia. It is in a location where epitheliomas are rather frequent. I am sure it will respond to proper treatment with roentgen rays, and one can be reasonably certain of the diagnosis on clinical grounds alone of morphea-like epithelioma.

DR. E. W. ABRAMOWITZ: I think that the condition is a morphea-like basal cell epithelioma, but I do not agree with Dr. Wolf's recommendations for treatment. I think that the fibrous element present requires destruction with electrocoagulation.

DR. GEORGE M. LEWIS: I too agree with the diagnosis of morphea-like epithelioma, but I should like to suggest a third method of treatment. Lesions of this type are usually radioresistant. After electrodesiccation the site of the lesion will show a depressed scar. In order to obtain the best results in this case I favor either simple excision or excision with plastic surgery.

DR. CHARLES WOLF: If this lesion is not properly treated with roentgen rays, the result will be poor. On the other hand, if one gives the proper dose of unfiltered roentgen rays, 4,500 r, at one sitting, there will be no vestige of the former lesion. An article by W. S. MacComb (*Am. J. Roentgenol.* **41**:437 [March] 1939) reports a series of 50 cases, in 95 per cent of which a cure was obtained after immense doses of roentgen rays which dermatologists are unaccustomed to administer.

DR. FRED WISE: What are the ultimate sequelae of a single dose of 12 skin units of roentgen rays?

DR. CHARLES WOLF: There is an epithelitis for two weeks and then it clears up. There are no ill effects. Today, with the improved technic, the sequelae one would expect to see do not develop.

DR. EUGENE F. TRAUB: The old Sherwell method of using the curet and applying 40 to 60 per cent acid mercuric nitrate to the base, if it is done properly, is an excellent method of treatment for this type of growth. It leaves no deformity and no telangiectasis and avoids all the controversy about irradiation.

DR. HERMAN GOODMAN: I hoped that some one might suggest morphea as a diagnosis. At one time I thought of morphea because of the clinical appearance.

NOTE.—The patient after this presentation went to the Memorial Hospital. A piece of tissue removed was reported as basal cell epithelioma.

A Case for Diagnosis (Lupus Erythematosus, Chronic Disseminate Type? Leioderma Syphiliticum?). Presented by DR. E. W. ABRAMOWITZ.

Miss J. G., a Negress aged 38, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, with an eruption involving the face and trunk of nine years' duration. The patient has irregular, well defined, circinate, oval chronic lesions on the face, with depigmented centers and darker peripheries. Similar lesions have developed on the lower part of the back in the past year and also on the knees and the forearms. She has received many injections of bismuth and gold compounds, with some improvement.

The Wassermann and Kahn reactions were negative on Feb. 24, 1937. Dr. Satenstein stated the belief that the histologic picture was that of lupus erythematosus.

DISCUSSION

Dr. E. W. Abramowitz: The first biopsy specimen, taken about two years ago from a lesion on the face, showed no sign of lupus erythematosus microscopically. It was suggestive of syphilis. The appearance of the second biopsy specimen, taker later from another lesion on the face, was definitely in favor of lupus erythematosus. She received injections of a gold compound with practically no improvement. Then she was given a bismuth compound, and the facial lesions showed some improvement. However, during the treatment with bismuth, an eruption developed on the back. There is no clinical history of syphilis. She has had two negative Wassermann reactions. I am not satisfied that she has lupus erythematosus. I do not want to introduce any new terms into syphilology, but Dr. Oppenheim, of Vienna, who observed the patient at the clinic, suggested the term leioderma syphiliticum for the lesions (if they are syphilitic), to describe the superficial smooth type of eruption serpiginous in outline and scar forming.

A Case for Diagnosis (Prurigo Nodularis?). Presented by Dr. James L. Miller.

M. L., a Negress aged 27, is presented from Vanderbilt Clinic, with a generalized eruption of sixteen years' duration. The patient has been studied in various hospitals in Pennsylvania and has apparently had the same type of eruption for the entire period of sixteen years. The eruption began at the age of 12 and first occurred on the extensor surfaces of the arms and on the front and back of both legs. It developed as a rash which consisted of many small "bumps" that itched. The condition gradually spread over the entire body. At the present time the skin is dry. Some areas are lichenified, and there are numerous small nodules. In some areas there is scaling, and in others, hyperpigmentation. At the site of removal of tissue for a biopsy there is a keloid. The injection of strontium bromide intramuscularly gave temporary results. The patient has had many types of therapy and states that she has improved slightly for a short time and has then reverted to her original state.

The Wassermann reaction has been repeatedly negative. Blood count showed 76 per cent hemoglobin, 3,720,000 red blood cells per cubic millimeter and 4,800 white blood cells per cubic millimeter, with a differential count of 50 per cent polymorphonuclear leukocytes, 34 per cent small and 2 per cent large lymphocytes, 6 per cent monocytes and 8 per cent eosinophils. There was slight anisocytosis. Histologic examination showed a picture of chronic dermatitis. The sections showed evidence of hyperkeratosis and acanthosis. Much melanin was seen in the epidermis. This was not entirely confined to the basal layer. In the corium there were moderate perivascular edema and infiltration of mononuclear cells.

DISCUSSION

Dr. E. W. Abramowitz: Are there any signs of chronic lymphatic leukemia?

Dr. James L. Miller: The results of physical examination have been entirely negative.

Dr. Louis Tulipan: I hesitate to accept the diagnosis of prurigo nodularis in a girl of that age, especially because the condition started when she was 12. Prurigo nodularis usually occurs in middle-aged women. Then, too, these lesions are really not the nodules of prurigo nodularis but papules with scratched tops. Nodules of prurigo nodularis are usually larger, more infiltrated and more fibrous, and they have keratotic surfaces. I believe that the condition is just prurigo that belongs in the papulourticarial group and should not be labeled prurigo nodularis.

A Case for Diagnosis (Hemangioendotheliosis? Majocchi's Disease? Kaposi's Idiopathic Hemorrhagic Sarcoma?). Presented by DR. ANDREW H. MONTGOMERY.

S. P., a man aged 30, is presented from the New York Polyclinic Hospital, with an eruption involving only the feet and legs and of indefinite duration. He is a weaver by occupation and has been blind from birth. He is growing increasingly deaf. He presents angiomatous and telangiectatic lesions and scars and various-sized atrophic patches. The more recent eruption is limited to the right shin and the inner toes of both feet. Some of the scarring is probably due to traumatism caused by his blindness. The patient first appeared at the hospital six months ago. Since then the condition has not changed appreciably except that most of the more elevated angiomatous lesions have become flatter. Subjective symptoms are negligible. He has had no local treatment except to injuries about the left ankle during this time. He had no treatment elsewhere previously.

On the right shin is a palm-sized, thinly keratinized patch through which telangiectasis of the deeper cutaneous vessels is visible. In its upper part there

Fig. 2.—A case for diagnosis (hemangioendotheliosis? Majocchi's disease? Kaposi's idiopathic hemorrhagic sarcoma?).

is a scar due to trauma. On the dorsum of the big and second toes of both feet and on the sides of the first and second toe spaces there are angiomatous and telangiectatic patches bordering atrophic areas. There are numerous pigmented or pale small patches of atrophy on the dorsolateral aspects of the feet. Below the malleoli are somewhat diffuse purpuric, atrophic and scaly areas. There is no associated varicose vein complex. While the condition of the eyes, ears and teeth suggests congenital syphilis, the Wassermann reaction of the patient's blood and that of his mother's blood were negative. Other observations on the blood and the basal metabolic rate have been normal.

Histologic examination of a lesion on the dorsum of the right big toe was made by a general pathologist and by two dermatologists. The general pathologist's report was as follows: The section showed some thinning and slight scaliness of the epidermis, with moderate atrophy of the papillary layer. There was a marked hyperplasia of the adventitial cells, encroaching on and narrowing the lumens of the small vessels, with telangiectasia. Little cellular inflammatory reaction was noted. The condition simulated an endarteritis that

was more truly a periadventitial hyperplasia. There were scattered areas of pigmentation that might be of hematogenous origin. The diagnosis is purpura annularis telangiectoides.

The report of a dermatologist was as follows: The epidermis of these sections showed a marked hyperkeratosis and parakeratosis. Acanthosis had also occurred. The corium showed a lobulated angiomatous proliferation. The caliber of the vascular channels was capillary or precapillary. The walls were thickened, apparently because of the proliferation of endothelial-like cells. There was hyalinization of the walls of the vessels in places. Hemorrhage and deposition of hemosiderin were noted. It was difficult to decide whether to consider this angiomatous tumefaction of the corium to be Majocchi's disease or a flat type of Kaposi's sarcoma.

The third report, by another dermatologist, was as follows: The section showed a form of vascular neoplasm, an endothelioma and perithelioma, with angiomatous development, new blood vessel formation and various stages of vascularization. It was considered that the diagnosis was not Kaposi's sarcoma or Majocchi's disease.

DISCUSSION

DR. FRED WISE: From the clinical standpoint I should eliminate consideration of Kaposi's sarcoma and Majocchi's disease. I do not think the condition resembles either of those conditions. On superficial examination, the resemblance to pernio is greater than to either of the two diseases mentioned. I should like to call your attention to publications in the French literature, especially to a series of articles by Gougerot and collaborators, in which is described a series of different types of eruptions due to capillary abnormalities. They called the eruptions capillaritis. I believe that the condition in this case belongs to such a group. It would be a good idea to perform a biopsy and use the method of capillaromicroscopy to see in precisely which category of the French group of capillaritis the condition in this case belongs.

DR. EUGENE F. TRAUB: In informal discussion I mentioned the possibility of frostbite or pernio. Considering this man's occupation and physical handicaps, I do not know why this might not be a very simple explanation for his condition. He does not know what happens to him most of the time, since he is blind and deaf, and he might easily have been frostbitten. That might also account for the ulcers that occur from time to time.

DR. ANDREW H. MONTGOMERY: I had not considered pernio. I had thought of the condition Dr. Wise mentioned, a lichenoid capillaritis, but it seems to be a much more superficial condition than that in this case. There is a large amount of atrophy here, and the condition is rather deeply seated.

Pigmented Nevus with Involvement of the Sclera. Presented by DR. ANDREW H. MONTGOMERY.

A. C., a girl aged 17, is presented from the New York Polyclinic Hospital with a lesion involving the right eye, present since birth. The patient presents a diffuse grayish blue pigmentation about the right orbital and temporal regions. It is most evident in the central portion. Over the eyelids there is a homogeneous pigmentation. The border is rather diffuse and patchy. The sclera has a deep purple pigmentation. The conjunctiva is normal except at the border, where there are some areas of pigmentation.

Vision is normal (right eye, 20/50; left eye, 20/20). The fundi are normal except that the one on the right is darker than that on the left.

Synovial Cyst of the Skin. Presented by DR. EUGENE F. TRAUB.

M. L., a woman aged 62, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital (patient of Dr. Harry

Keil). About one year ago on the left middle finger there appeared a small lesion resembling a "corn," which has been associated for the past few months with fluid. Several days ago the patient used some irritating cleansing fluid, and there appeared some redness of the lesion accompanied by slight pain.

There is a lesion slightly smaller than a dime on the outer aspect of the left middle finger near the distal interphalangeal joint. This is a cystlike structure with thick horny covering, through which, however, the underlying clear contents can be seen. The periphery is reddish brown, apparently because of local irritation. Pressure on the lesion causes some pain.

EUGENE F. TRAUB, M.D., *Chairman*

May 2, 1939

LOUIS TULIPAN, M.D., *Secretary*

Atrophoderma. Presented by DR. OSCAR L. LEVIN.

I. L., a woman aged 18, born in Poland, is presented from the Beth Israel Hospital with an eruption involving the upper extremities, of about nine months' duration. The patient appeared at the hospital complaining of an eruption in the axillas which was diagnosed as dermatitis venenata. In the course of examination several pea-sized violaceous soft masses which appeared to be movable with the skin were noted. These lesions were discrete and were located on the extensor surfaces of the forearms and arms. There was no subjective sensation. One of the lesions on the right forearm showed evidence of spontaneous resolution, and at the site of the lesion there was superficial atrophy.

Histologic examination revealed the horny layer to be moderately thick. There was no parakeratosis. The stratum granulosum was thick, the stratum spinosum consisted on the average of six layers and the basal layer was indistinct. It was moderately pigmented. The corpus papillare was mostly obliterated. Under the epithelium there was a thin layer of rather fine collagenous fibers, mostly running parallel to the surface. In this layer elastic lamellae appeared numerous, mostly thin, broken up and in multifarious fashion beaded, looped, club shaped and irregularly thickened. The elastic tissue in the remnants of the corpus papillare was fairly normal. The cutis had normal thick collagenous fibers and somewhat decreased elastic tissue with less degenerative change. There was one normal-appearing sebaceous gland and hair follicle. Only a few rather thin coils of sweat glands were seen. There was one normal arrector muscle. Occasionally melanotic pigment was found in large connective tissue cells not far from the epithelium. In the superficial layers of the connective tissue the blood vessels were surrounded by loose aggregations of mononuclear elements.

Lichen Planus Sclerosus et Atrophicus (Hallopeau). Presented by DR. JESSE A. TOLMACH.

M. E., a woman aged 35, born in Austria, a housewife, is presented from the Beth Israel Hospital with an eruption of eighteen months' duration involving the chest and the upper part of the back. The eruption began with the appearance of small, flat, whitish, irregularly shaped hard lichenoid papules. Some of these showed central black horny follicular plugs. Large round and irregular ivory-colored hard patches soon developed, with areas of hyperpigmentation at the peripheries. Central black follicular plugs were noted in the large lesions. The lesions have been showing a tendency to spontaneous resolution with atrophy.

Histologic examination by Dr. Else A. Barthel showed an atrophic epidermis with swelling of some of the basal cells and a nonparakeratotic scale. The numerous dilated hair follicle openings were filled with horny plugs. There was a band of swollen and degenerated collagen fibers in the upper third of the corium.

The normal structures were absent. Whatever blood vessels were present, especially at the lower level of the involved area, were large spaces. Nuclei were absent except just below this level, where they made a thin, bandlike line. It gave the impression of the remains of an inflammatory exudate. The Weigert stain showed an absence of elastic tissue fibers in the upper layers of the corium and a swelling of the fibers at the level of the line of nuclei. Although the main changes were morphea-like, it was the conclusion that the dilated hair follicles with their horny plugs and the remains of an inflammatory exudate at the base of the lesion have to be considered as part of a previous activity, and therefore a diagnosis of lichen sclerosus or lichen albus von Zumbusch is justified. The Wassermann, Kline and Kahn reactions were negative. Urinalysis gave negative results.

DISCUSSION

DR. FRED WISE: In my opinion it is best to eliminate the term "lichen planus" in discussing an eruption which is devoid of the lesions of lichen planus either at the time the patient is observed or at some previous time during the course of the eruption. Clinically some of the lesions bear only a superficial resemblance to genuine lichen planus. Although the designation "dermatitis lichenoides chronica atrophicans" is somewhat cumbersome, it seems to identify the true character of the eruption and avoids confusion with lichen planus atrophicans, a different disease.

"White spot disease" is recognized as scleroderma occurring in small isolated areas—morphoea guttata. In this disease the elastic tissue is retained, while in the late stages of chronic lichenoid dermatitis the elastica is fragmented and greatly diminished or entirely destroyed. White spot disease occurs most frequently on the subclavicular, midsternal and deltoid regions and sometimes on the genitalia (MacKee, G. M., and Wise, F.: White Spot Disease, *J. Cutan Dis.* **32:** 529, 1914).

DR. JESSE A. TOLMACH: I agree with those who believe that this type of eruption should be called lichen sclerosus et atrophicus rather than lichen planus sclerosus et atrophicus, because neither from the history nor from the examination is there any evidence of lichen planus. The presence of black horny follicular plugs in the centers of morphea-like lesions is typical of lichen sclerosus et atrophicus. This condition apparently lies somewhere between lichen planus atrophicus and morphea. It is probably a variant of morphea.

Dermatophytosis. Presented by DR. JESSE A. TOLMACH.

A. F., a man aged 36, born in Hungary, resident three months in the United States, is presented from the Beth Israel Hospital. The patient states that he has an eruption of more than four years' duration. It was treated in Vienna, Austria, yielded only temporarily to therapy and recurred shortly after treatment was discontinued. The eruption is extensive, involving large areas of the trunk, extremities, axillas and genitocrural region. It consists of large patches, sharp in outline, with arched and polycyclic borders. It is erythematous and scaly. Papulovesicular lesions are scattered throughout the patches. The skin is not infiltrated to any extent, only the margins being slightly elevated. There are numerous excoriations due to severe itching and scratching.

The Wassermann reaction was negative. Urinalysis gave negative results. The blood count showed 5,210,000 red blood cells, with 82 per cent hemoglobin, and 9,600 white blood cells, with a differential count of 57 per cent polymorphonuclear leukocytes, 35 per cent lymphocytes, 6 per cent monocytes, 1 per cent eosinophils and 1 per cent basophils. Examination of the scales for fungi gave positive results. Cultures were made but have not yet been reported on. Histologic examination showed a productive inflammation around the superficial blood vessels of the skin.

The patient is in good health except for the cutaneous condition. He underwent a tonsillectomy in September 1938. Treatment has consisted of application

of compound ointment of benzoic acid N. F. (Whitfield's ointment) to a patch on the skin of the abdomen. This produced complete disappearance of the eruption in three days.

DISCUSSION

DR. FRED WISE: The eruption at present bears a close resemblance to dermatitis herpetiformis except for the lesions on the perigenital areas, which obviously are caused by Epidermophyton inguinale. It would interest me to learn what organism has been isolated from the lesions on the trunk.

DR. SAMUEL M. PECK: This case is of extreme interest. It is important to know whether a trichophytin test was made. A number of years ago I observed a case in which the patient was covered practically from head to foot with one solid superficial scaly patch of dermatophytosis. It was easy to demonstrate the fungus in cultures of material from the advancing border. There was a tendency for the condition to clear up spontaneously toward the center of this lesion. The trichophytin test is most important because it can be used to determine whether the condition is an unusual dermatophytosis or a dermatophytid. It must be realized that even in dermatophytosis it is possible occasionally to find fungi. If the result of the trichophytin test were negative, one could assume more readily that the condition is dermatophytosis without any allergic manifestations. It is important also to make cultures in this case because it is hard to conceive that Trichophyton gypseum, for instance, should give such a widespread eruption without the development of hypersensitivity. However, an organism like Trichophyton purpureum might possibly give such a widespread infection without any associated sensitivity to the fungus. From the appearance of the lesion it is most likely that the condition is Duhring's disease with associated dermatophytosis in the groin.

DR. GEORGE M. LEWIS: There is little doubt that this man has dermatophytosis affecting the interdigital webs of the feet and the inner aspects of the thighs. Was the examination for fungi made on a lesion from one of those areas?

DR. JESSE A. TOLMACH: The fungi were found in lesions from the abdomen.

DR. GEORGE M. LEWIS: The eruption on the body, as Dr. Wise has suggested, bears a striking resemblance to dermatitis herpetiformis, and I favor that diagnosis. The symmetry of the lesions, for one thing, is against tinea. The character of the lesions also is against this diagnosis. If the cultures of material taken from the truncal lesions are sterile or contaminated, the organism demonstrated in the direct mount may have been a saprophyte. If the lesions on the trunk are of mycotic origin, the most likely pathogen is T. purpureum. The result of the trichophytin test would be interesting, as Dr. Peck has brought out, particularly if a wheal reaction were noticed after ten minutes. That would favor the diagnosis of an infection due to T. purpureum.

DR. HERMAN SHARLIT: It appears to me that it is not yet justifiable to accept this case as one of extensive tinea infection, in spite of the fact that scrapings from the lesions on the trunk show fungi. One may readily concede that the patient has tinea in the groin and on the feet, but there are features so strikingly suggestive of Duhring's disease that one cannot relinquish this possibility until a report is available indicating that the fungi on the trunk are pathogens. If this turns out to be the case, of course tinea is the diagnosis. If the diagnosis of tinea cannot be maintained on the basis of laboratory findings, the presence of the prefungoid stage of mycosis fungoides should be considered.

DR. R. H. RULISON: I saw this patient but unfortunately did not see the case history. I am always slow to accept a positive laboratory finding as sufficient to explain a condition. In this case every one seems to agree that a ringworm infection of the toes and groins is present. The eruption on the rest of the body strongly suggests Duhring's disease. I see no reason why both conditions may not be present, and the finding of fungi in scrapings from lesions on the abdomen does not warrant the opinion that the entire process is a ringworm infection.

DR. FRED WISE: I should like to be informed as to the clinical differentiation of eruptions on the trunk caused by the purpureum fungus and eruptions caused by the gypseum fungus. I recall the case of "purpureum sycosis" of the beard, reported several years ago by Lewis and Harris, in which the condition clinically resembled tinea profunda. Can the eruptions caused by these two forms of fungus be differentiated on clinical grounds alone?

DR. GEORGE M. LEWIS: There are certain features of an infection due to T. purpureum which are fairly distinctive. Students at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital frequently make the correct diagnosis from inspection alone. Further observations have confirmed the conclusions reached in our report (ARCH. DERMAT. & SYPH. **37**:823 [May] 1938).

DR. E. WILLIAM ABRAMOWITZ: Infections due to T. purpureum are exceedingly resistant to treatment.

DR. EUGENE F. TRAUB: The application of compound ointment of benzoic acid to some of the lesions caused their complete disappearance, according to the history.

DR. HERMAN GOODMAN: I should like to ask two questions. One is whether this case as we have observed it tonight and the description given by Dr. Lewis would lead Dr. Wise to say that this is a case of infection due to T. purpureum? It seems to me that many of the lesions described are not observed in the patient as presented. This man shows enough vesiculation to justify a diagnosis of Duhring's disease. Secondly, is the reaction to trichophytin which occurs within ten minutes specific or nonspecific? Would the man react in ten minutes to an injection of typhoid vaccine or other substances?

DR. EUGENE F. TRAUB: I should also like to ask Dr. Lewis a question. He stated that the most common infection of the nails is due to T. purpureum. Does he mean that the most common form of fungous infection affecting the feet is that due to this organism? I ask this because in most cases of fungous infection of the foot the nails also are affected.

DR. GEORGE M. LEWIS: I always control the injection of trichophytin by injecting, in addition, saline solution and oidiomycin. There is no doubt that trichophytin elicits a specific reaction after forty-eight hours, and I believe the immediate wheal reaction to be equally specific. T. purpureum is responsible in the majority of cases for onychomycosis in which there is deep invasion of the nail. T. gypseum is a frequent finding on the surface of nails which show little evidence of destruction or invasion and is important simply as a focus.

DR. JESSE A. TOLMACH: All the discussion of this case has been based on the assumption that it is a case of proved dermatophytosis. It is interesting to realize that dermatophytosis may closely simulate Duhring's disease or mycosis fungoides. Our first diagnosis in this case was Duhring's disease. The histologic report suggested the possibility of prefungoid mycosis fungoides. A few days ago I scraped several areas on the abdomen and on microscopic examination found mycelial threads. The culture has not yet been reported on. I believe it will be necessary to postpone definitely diagnosing this eruption as dermatophytosis until we are able to confirm the microscopic finding with a culture yielding a pathogenic fungus. All soothing local applications had previously not improved the eruption. After finding the mycelial threads microscopically, we applied compound ointment of benzoic acid to the patches on the abdomen and within two days noted a marked improvement. This, too, tends to support the possibility of a fungous infection.

Lupus Erythematosus (Epithelioma of the Lip?). Presented by DR. EUGENE F. TRAUB.

M. T., a man aged 63, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital with an eruption of twenty-six years' duration involving the face, ears and lip. He was admitted to the hospital

in August 1921 and received injections of a gold compound. The right ear and the adjacent skin and scalp are the site of a plastic operation. There is no evidence of lupus erythematosus in this region at present. The left ear shows atrophy of the lobe and of the skin around the lower part. An epithelioma was removed from the scalp in April 1924. In 1928 a lesion occurred on the right ear. This was removed, the removal being followed by a plastic operation. The eruption on the face is erythematous and scaly, with some atrophy on the left side of the chin, extending to the right side of the upper lip. There is an ulceration of eight months' duration, about 1 cm. in diameter, on the right side of the lower lip. This presents some induration. In July 1937 a V-shaped wedge was removed from the left side of the lower lip; this microscopically showed inflammatory induration.

The case is presented for opinion regarding therapy and diagnosis of the ulceration on the right side of the lower lip.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: As in lupus vulgaris, malignant change can develop in long-standing lupus erythematosus, irrespective of whether the patient has received radiotherapy. I should like to know what treatment the patient received.

DR. EUGENE F. TRAUB: The patient has had plenty of treatment with gold and bismuth compounds and also considerable roentgen therapy.

DR. E. W. ABRAMOWITZ: Even when the patient fails to respond to gold or bismuth compounds, local stimulation—for instance, with a mild application of the cryocautery—sometimes produces results. If both methods fail, the patient should be given sulfanilamide in 5 grain (0.32 Gm.) doses four times daily.

DR. ISADORE ROSEN: In addition to what Dr. Abramowitz has said, I think the first thing to do in this case is to determine by biopsy whether the patient has an epithelioma. The section should be removed from the suspicious-looking area. There is no doubt that the man has lupus erythematosus; the question is whether this is undergoing malignant change.

DR. JESSE A. TOLMACH: About a year ago I presented a young man with lupus erythematosus of the ulcerative type. The ulcers resembled gummas. Although I agree, of course, that a biopsy should be performed in this case, it should not be forgotten that there is an ulcerative type of lupus erythematous. In the case I have mentioned, the ulcers all closed up under treatment with injections of a bismuth compound and administration of potassium iodide by mouth. I therefore suggest that form of treatment in this case.

DR. EUGENE F. TRAUB: The only thing I can add about treatment is that the patient has had a considerable amount of roentgen ray treatment about the ears. He had none on the chin or lip. He has had a great deal of treatment with a gold compound and a certain amount of autogenous vaccine therapy.

A Case for Diagnosis (Seborrhea-like Eruption). Presented by DR. HERMAN GOODMAN.

S. B., a man aged 52, is presented for Dr. Brunor. The patient has always been well and never had reason to visit a physician. The only previous eruption occurred at the age of 8 or 9 years. This eruption was diagnosed as "mange" acquired from a dog. He states that he has never had syphilis or gonorrhea or any symptoms of either disease. About five or six months ago he noticed a small "sore" on the back of his neck. He has no knowledge of how it began. He may have picked at the sore. He applied phenolated petrolatum. The eruption spread over the entire scalp and then involved the face. Two weeks after the first appearance of the sore on the back of the neck, the scalp and face showed many pustules and vesicles which oozed and formed crusts. After this the face became very red. He stopped using phenolated petrolatum. Four months later he went to a physician, who

removed hair from the scalp and applied zinc oxide ointment. Injections were made into the blood. There was no improvement. He then used coconut oil, which seemed to cause a spread of the eruption. The next change was to linseed oil, without improvement. For the past six weeks he has been under a physician's care and has had a widespread eruption involving the arms, the shoulders, part of the chest, the legs and the buttocks. The nose has always been clear, as have the palms. The patient has been given sodium thiosulfate baths and injections of sodium thiosulfate as well as calcium bromide, without any relief.

The patient is a bookkeeper in a commission house selling fish. He lives at the home of his brother-in-law. He is not exposed to any known fumes or noxious substances and has not changed his mode of living for six years. The apartment in which he lives and the office in which he works have not been painted for a year or more. There are no plants in the house. He has no recollection of any change in his tailor or in his tonsorial procedures prior to the onset of the present eruption.

The scalp, face and upper part of the body are red, scaly and partially infiltrated. There are no clear areas of skin in these regions. The rest of the body presents widespread discrete areas of an eruption which is red, partially infiltrated and scaly. The inner sides of the arms and forearms about the cubital fossae present wide areas of lichenification. The nails show transverse lines close to the cuticle. There is slight thickening of the skin between the toes, especially the small toes. The patient feels cold on exposure of the body to the temperature of a fairly warm room. He smokes cigarets but rarely touches liquor of any kind. He had not taken any medicines internally or used any externally prior to the present eruption. The legs have been swollen for about one week. The urine was reported as free of albumin or casts. The Wassermann reaction was negative.

This patient is presented for suggestions as to ambulatory treatment, since he will lose his position if he stays away from work.

DISCUSSION

DR. FRED WISE: The eruption appears to be either psoriasis or seborrheic eczema. Possibly it is a mixture of the two.

DR. E. WILLIAM ABRAMOWITZ: I agree with the diagnosis offered by Dr. Wise. In view of the fact that the condition has not responded to the usual methods, I should like to suggest 30 per cent sulfur in petrolatum.

DR. PAUL GROSS: I agree with Dr. Wise.

DR. CHARLES WOLF: From the casual examination that we all made, I should like to offer the diagnosis of exfoliative dermatitis, which perhaps started as an infection, possibly as tinea. It would also be wise to rule out a generalized dermatophytosis. I should therefore make a tentative diagnosis of exfoliative dermatitis due to medication, or a disseminated dermatophytosis.

DR. EUGENE F. TRAUB: I agree with Dr. Wise. I do not know how one can choose between the two diagnoses suggested without a histologic examination, but I favor the diagnosis of seborrheic dermatitis.

DR. HERMAN GOODMAN: We will try 30 per cent sulfur in petrolatum according to the prescription of Dr. Abramowitz, exactly as he uses it.

Morphea-Like Epithelioma of Nasolabial Fold. Presented by DR. HERMAN GOODMAN.

D. L., a woman aged 37, was presented on April 4, 1939. There was a question as to whether the lesion was morphea-like epithelioma or morphea. The patient went to the Memorial Hospital, where a section was removed. It was reported as basal cell epithelioma, and the patient is now being treated with fractional doses of roentgen rays, unfiltered, at weekly intervals, according to the routine technic used at that hospital.

MANHATTAN DERMATOLOGIC SOCIETY

MAX SCHEER, M.D., *President*

April 11, 1939

ANTHONY C. CIPOLLARO, M.D., *Secretary*

Tuberculous Ulceration of the Nose. Presented by DR. MAURICE J. COSTELLO.

M. F., a woman teacher aged 60, has had an extensive ulceration of both nasal cavities, with complete destruction of the lower half of the septum, since November 1937, one and a half years. She was presented before this society in February 1938 (ARCH. DERMAT. & SYPH. **38**:106 [July] 1938). At that time the lesion had healed.

The ulceration of the nose recurred nine months later and again failed to respond to antisyphilitic therapy, including administration of iodides. There was some improvement with the administration of neoprontosil in large doses.

Last Friday the patient was seen in consultation with Dr. Joseph Kelly, an otolaryngologist, who believed that the condition was unquestionably tuberculosis and advised the resumption of the tuberculin therapy. On that day she was tested on the left arm with a 1:10,000,000 dilution of old tuberculin and on the right arm with dilution of 1,100,000,000 and 1:1,000,000,000. The reaction was similar to the reactions previously reported, although not quite so violent. There have been local, focal and constitutional reactions. It is believed that the patient had a tuberculous ulceration of the nose, which is a local violent response in an extremely allergic patient. Tuberculin therapy will be continued.

DISCUSSION

DR. GEORGE M. LEWIS: The reactions are severe enough to simulate inoculation tuberculosis. In the latter condition, however, the tumefaction is deeper, and there is little tendency to spontaneous disappearance.

DR. MAX SCHEER: Regarding the resistance, I think this decided reaction to tuberculin is an evidence of high resistance to injected tuberculin rather than of low resistance.

DR. E. WILLIAM ABRAMOWITZ: I am well aware that reactions to tuberculin are usually specific. Reactions in this patient are rather paradoxic, those to low and to high dilutions being practically the same. This question evidently bothered the members at the time of the previous presentation, because of a possible iodine sensitization. One must remember that tuberculin has produced beneficial results in other conditions besides tuberculosis (for instance, acne and dementia paralytica). I think the improvement in this patient following tuberculin therapy may be due to nonspecific effects of the drug.

DR. DAVID BLOOM: I am wondering whether this decided sensitivity to tuberculin is not the explanation for the nonspecific tissue found on pathologic examination. The tuberculin reaction is the only definite point in favor of the diagnosis of tuberculosis. Animal inoculation and particularly investigation along the lines given by Mellon (ARCH. DERMAT. & SYPH. **37**:451 [March] 1938) may be fruitful in proving the diagnosis.

DR. MAURICE J. COSTELLO: Two weeks after the injections of tuberculin were made, the condition had disappeared, and the patient remained free of symptoms for nine months. It was not determined at that time whether the improvement was the response of a tertiary syphilitic lesion to potassium iodide therapy or whether it was due to the tuberculin. When the condition recurred, she was again treated with injections of liver extract and antisyphilitic drugs, without benefit. She was tested with tuberculin again and told that she would probably have a severe reac-

tion. The dilutions were increased. There has been some difference in the reactions. Now she has a local, a focal and a constitutional reaction. These facts, with the information that she also has a healed tuberculosis of the lungs and that she had a papulonecrotic tuberculid, add weight to the diagnosis of tuberculous ulceration of the nose, even though a tuberculoid structure was not observed histologically.

Steatocystoma Multiplex Resembling Acne Vulgaris. Presented by DR. MAURICE J. COSTELLO.

J. A., a boy aged 15½, has been treated for hypopituitarism and hypothyroidism for several years. The eruption he presents has been present for two months. It consists of numerous discrete, diffuse pinhead-sized to buckshot-sized elevated lesions, which have eccentrically placed follicular openings, through which sebaceous material can be expressed. He also has a number of spinous processes on the posterolateral aspect of the arms and the posterior axillary borders. The eruption involves the skin covering the chest, the back and the shoulders, roughly, the acne areas. He has been taking thyroid tablets for some time. He had grip and otitis media several months ago, about the time of the onset of this eruption.

DISCUSSION

DR. FRED WISE: I do not agree with the presenter's diagnosis. The lesions are much too small and are confined to the hair follicles; they do not contain any visible pus, as do lesions in most cases of multiple steatomas. I am more inclined toward the diagnosis of keratosis pilaris. This patient should get well under vitamin A therapy, although I was disappointed in the treatment of a patient with keratosis pilaris, to whom large doses of halibut liver oil were administered.

DR. GEORGE C. ANDREWS: I am very skeptical as to the value of vitamin A in the treatment of this patient or of any other patient with a cutaneous disease who lives in the city of New York. It is doubtful whether deficiency of vitamin A exists in New York. This is a case of multiple steatomas of an unusually small type, associated with a glandular condition, both hypopituitarism and hypothyroidism. In some cases of multiple steatomas there is an increased amount of cholesterol in the blood, and of course sometimes the steatomas are familial. Glandular treatment would be helpful.

DR. WILBERT SACHS: The lesions of this patient do not look like the lesions of any of the patients whose cases I have reported. These lesions are small, uniform in size and skin colored. In my patients the lesions were present chiefly on the face, the back and the scalp, varied in size from that of a pinhead to that of a pea and were skin colored, yellow or brown. There were no subjective symptoms, and many of the lesions became infected. I do not recall just what the cholesterol content of the blood was. In the cases reported in the literature, the lesions were present for many years, but in my cases they were present at birth.

DR. E. WILLIAM ABRAMOWITZ: I doubt whether the general condition should be accepted as a true Fröhlich syndrome, because the boy has a pretty good development of the genitals and the other sex characteristics are normal. The boy has a cretin-like facies, a dry skin and keratosis pilaris, part of the picture of hypothyroidism. I do not know whether the other cutaneous lesions are true steatomas or not.

DR. MAX SCHEER: I am inclined to agree with Dr. Abramowitz, because the boy does not have all the features of Fröhlich's syndrome. He has normal-sized genitals.

DR. MAURICE J. COSTELLO: This boy has been under endocrine treatment for some time, and I imagine the clinical picture, as far as the Fröhlich syndrome is concerned, might have been changed. The lesions were incised, and a liquid type of material was expressed, different from the material expressed from ordinary acne lesions or comedos, and much like the material expressed from multiple

steatomas. It is possible, too, that his diet is pretty well restricted to fatty foods, and he may not get the normal amount of cream, yolk of eggs and vitamin A.

A Case for Diagnosis (Lupus Erythematosus Bullosus?). Presented by Dr. Paul E. Bechet.

E. M., a woman aged 49, was presented by Dr. Fred Wise before the New York Dermatological Society on March 28, 1939 (Arch Dermat. & Syph., this issue, p. 1030) with a condition to be diagnosed. The tentative diagnosis was dermatitis herpetiformis.

DISCUSSION

Dr. Isadore Rosen: The clinical features that this patient presents, i. e., grouped vesicobullous lesions, occurring in crops and although limited to the face and neck, persisting for many months, suggest the diagnosis of dermatitis herpetiformis. That the lesions are so localized does not rule out that diagnosis, for there are many atypical forms of dermatitis herpetiformis, just as there are atypical forms of other cutaneous diseases.

Dr. Max Scheer: As Dr. Rosen brought out, there are unusual varieties of diseases, and there are also transitional forms between different diseases. There are cases in which it is impossible to distinguish psoriasis from seborrheic eczema, because of transitional forms, and I think there are such transitional forms between Duhring's disease and pemphigus. This patient has no itching, no grouping and no pigmentation. The distribution is unusual. While Duhring's disease is a possible diagnosis, I think other possibilities should be considered. I should like to ask Dr. Wise whether he has observed any cases or knows of any cases of Duhring's disease in which the diagnosis was confirmed histologically but the condition ultimately proved to be pemphigus.

Dr. Fred Wise: The microscopic differentiation between Duhring's disease and pemphigus is sometimes difficult, but the microscopic differentiation between Duhring's disease and lupus erythematosus pemphigoides is relatively simple; therefore, I think one should accept the histologic diagnosis submitted by Dr. Satenstein, i. e., Duhring's disease. In this particular instance the histologic diagnosis seems much more likely to be correct than the clinical one.

Dr. Mihran B. Parounagian: I am unwilling to accept the diagnosis of Duhring's disease in this case for the following reasons: because of the absence of itching, because of the limitation of the lesions to the upper portion of the chest for four years and because of the absence of the lesions in the characteristic locations, like the knees and buttocks. The suggestion of pemphigus is more acceptable to me.

Dr. Ludwig Oulmann: I want to recall a case which I presented here about one and a half years ago. The patient, a woman of over 70 years, had lesions which most of the members regarded as Duhring's disease, although I presented the case as one of pemphigus. The condition of this woman had improved at the time of presentation and showed a picture similar to that of the patient presented tonight. Nine months later she had a new outbreak, of a severe nature. She improved again but had a new attack three months ago, followed by pneumonia, to which she succumbed.

Dr. Paul E. Bechet: I cannot agree with the diagnosis of dermatitis herpetiformis. The clinical picture in my opinion does not even remotely suggest that diagnosis. The lesions are of five years' duration. There have been no remissions. During all that time they have been confined to the face, the arms and the upper part of the chest and back. The patient is fond of ocean bathing and states that she is distinctly worse after prolonged exposure to actinic rays. There is no grouping, scratch marks or pigmentation. The result of the patch test with an iodide was negative, and the eosinophil count was normal. With such clinical evidence, the diagnosis of a bullous type of lupus erythematosus is much more plausible than a diagnosis of dermatitis herpetiformis.

Lupus Vulgaris; Radiodermatitis. Presented by DR. GEORGE M. LEWIS.

L. Q., a man aged 56, first noted small reddish brown papules on the right temple in 1931. There was a gradual spread of the condition, and in 1932 the right cheek became involved. Later the rash affected the left cheek, the lower lip and the inner part of the cheek. A biopsy was performed in 1931 at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, and the diagnosis of lupus vulgaris was established. Previous therapy included the application of caustics; the administration of ultraviolet rays, roentgen rays and radium; a salt-free diet; intravenous injection of a gold preparation, and a plastic surgical operation. The patient has been treated in a number of local clinics and has also been in Vienna, Austria. He credits the Finsen lamp with the best results he has obtained. Recently, he has been given intravenous injections of gold sodium thiosulfate, and locally, to the ulcerated areas, fresh leaves of Aloe vera have been applied. Examination disclosed typical apple jelly nodules disseminated over the cheeks, the forehead and the eyelids. Lesions are present on the buccal mucosa. There is destruction of the right upper and lower eyelids. On the upper part of the right cheek, extending to cover part of the sclera, is a bright red, smooth localized elevation. There is severe pain in this region.

DISCUSSION

DR. FRED WISE: Dr. Lewis presented this patient for suggestions as to further therapy. I think the condition would be a difficult one to cure. I suggest, instead of applying Aloe vera leaves in an irregular manner, that the treatment should be given a thorough trial before any surgical method is instituted. I do not see any evidence of radiodermatitis, but I believe that the treatment with Aloe vera leaves is nevertheless indicated.

DR. DAVID BLOOM: As to surgical treatment, it is my opinion that as long as there is such an extensive active process surgical excision would be hardly possible.

DR. LUDWIG OULMANN: In May 1937 Goodman published directions for preparing an emulsion for use in cases of radiodermatitis (ARCH. DERMAT. & SYPH. **35**:942 [May] 1937). This emulsion might be beneficial in this case.

DR. GEORGE M. LEWIS: I hesitated at first to make a diagnosis of radiodermatitis because of the absence of telangiectasia; however, the severe and almost constant pain would favor that diagnosis for the ulcerated lesions. No improvement has been noticed from the injections of gold sodium thiosulfate; there have been lapses in the treatment by leaves of Aloe vera, and the salt-free diet has not been consistently adhered to. It is doubtful if surgical intervention is advisable until conservative measures have been tried a little longer.

Keratoderma (Arsenic?); Lupus Erythematosus with Epithelioma. Presented by DR. ISADORE ROSEN.

A. W., a man aged 65, came to the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on April 5, 1939, with an eruption of seven years' duration, which began on the left ear.

The patient now presents several areas on the cheeks and behind both ears, which consist of atrophic erythematous lesions having a rolled raised border, with infiltration and with nodular formation. Both palms show sharply marginated keratoderma. The patient gave a history of the ingestion of drops, probably solution of potassium arsenite U. S. P., and of injections for ringworm. He has also had six roentgen ray treatments.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: I should call the palmar lesion a diffuse keratoderma, the kind most likely to appear a short time after the use of arsenic. I should say that the patient has superficial epitheliomas of the right cheek and ears and a possible psoriasis of the palms.

DR. GEORGE C. ANDREWS: I saw this patient for only a moment, but I think the palmar lesions are characteristic of arsenical keratoses. There are hard black keratotic plugs which leave little pitted depressions when removed. These are not produced except by arsenical or roentgen ray or radium lesions. The lesion back of the ears is a superficial pagetoid type of epithelioma or Bowen's disease. The lesions on the cheek are typical epitheliomas.

Tuberculosis of the Tongue. Presented by DR. PAUL E. BECHET.

R. S., a man aged 47, was presented before this society in March 1939 (ARCH. DERMAT. & SYPH. **40**:623 [Oct.] 1939) for a diagnosis. He had come to the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on the day of the presentation, and no laboratory work, other than a dark field examination, had been done. The results of that examination were negative. The tentative diagnoses were chancre, tuberculosis and carcinoma.

The patient presents on the tip of the tongue a well defined lesion about 1 cm. in diameter, which has been present for two and a half months. It has an eroded sharply defined border and its floor is covered with a whitish yellow exudate. There is no adenopathy. The patient's general health is apparently good. He states he feels well, despite the fact that he has had a chronic cough of long standing, and for the last six months has had "night sweats."

The histologic diagnosis was tuberculosis cutis. Roentgenograms of the chest show a picture somewhat devoid of definite causative characteristics. Both a systemic and a lymphatic type of dyscrasia and also tuberculosis remain for clinical consideration as likely etiologic factors. Examination of the thorax showed the total heart area relatively a shade small, with, however, slight left ventricular and aortic prominence. There is a considerable degree of lymphoid nodulation at the hilus, with secondary root branch thickening, as well as further reticular and spotty lymphoid type peribronchial infiltration extending into the central lung fields well toward the periphery, a shade more discernible in the upper lobes.

DISCUSSION

DR. ANTHONY C. CIPOLLARO: I was much interested in this case, because usually orificial tuberculosis is accompanied by visceral tuberculosis. In this particular case there is no evidence of visceral tuberculosis. Another interesting thing is that in orificial tuberculosis the tuberculin reactions are usually negative. If this lesion is tuberculous, it certainly is a most unusual form.

DR. ISADORE ROSEN: In view of the fact that this patient has no clinical evidence of tuberculosis elsewhere, I do not see how a definite diagnosis of tuberculosis of the tongue can be made without demonstrating tubercle bacilli.

DR. WILBERT SACHS: I know that tuberculosis of the tongue is almost always secondary to visceral tuberculosis. However, this patient may not have any visceral tuberculosis; an inoculation tuberculosis may have developed from an external source.

DR. GEORGE C. ANDREWS: I think this patient should be hospitalized and studied carefully, because if the lesion is a focus of primary tuberculous infection it should be excised and removed. One probably would not get rid of all the tubercle bacilli that way, because they are probably already in the glands or further disseminated, but I think it is important that the primary focus be removed as quickly as possible. The exact diagnosis should be cleared up promptly.

DR. PAUL E. BECHET: The lesion has been present for three months. There is no adenopathy and no cutaneous eruption. The dark field examination gave negative results, and the Wassermann and Kahn reactions were negative. It would seem to me that this should exclude the diagnosis of syphilis. The histologic report states that the lesion is tuberculous. The patient has a chronic cough and night sweats, and the report of the roentgenogram of his chest, while couched in somewhat ambiguous language, states that tuberculosis may be a possibility. The

evidence in favor of tuberculosis, including the clinical appearance of the lesion, is therefore fairly strong. Despite the fact that the patient feels well and has not lost weight, I believe that there is sufficient clinical and pathologic evidence to make the diagnosis of tuberculosis reasonably certain.

Erythema Annulare Centrifugum (Darier). Presented by DR. GEORGE M. LEWIS.

G. L., an electrician aged 27, presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, first noticed an eruption on his trunk four months ago. This eruption has gradually increased in extent. He has not complained of itching.

He now has lesions of various size, chiefly on the left side of the trunk, across the upper part of the back and on the buttocks. All the lesions have clear centers and well marked erythematous borders. Some scaling is to be noted. Direct examination and cultural studies for fungi gave negative results on two occasions.

NOTE.—A subsequent histologic study was made by Drs. Satenstein and Sachs; their impressions were that there were changes suggestive of pityriasis rosea but not entirely substantial for that diagnosis.

DISCUSSION

DR. FRED WISE: I think the eruption is that of pityriasis rosea.

DR. WILBERT SACHS: We considered the possibility of pityriasis rosea. The lesions are mostly on one side of the body, of four months' duration, gyrate in appearance, with no cigaret paper wrinkling and no scaling on the edges, and therefore we considered these lesions as erythema annulare centrifugum. The lesions on the arm appeared recently and look like pityriasis rosea. It is possible for pityriasis rosea to develop in a patient who already has erythema annulare centrifugum.

DR. E. W. ABRAMOWITZ: Some of the members probably recall the article by Klauder (*J. A. M. A.* **82**:178 [Jan. 19] 1924), who described unusual types and locations of pityriasis rosea. Judging from some of the macular scaly lesions that the patient presents, the diagnosis of pityriasis rosea has to be eliminated.

DR. GEORGE M. LEWIS: The patient was first thought to present lesions of tinea of the smooth skin, and two examinations with negative results did not entirely rule out that diagnosis. The long duration, the absence of a herald patch, the elevated borders of the lesions and the lack of symmetry of the eruption serve to distinguish the disorder from pityriasis rosea. The lesions thought to show some resemblance to pityriasis rosea were freshly scraped, and this no doubt was confusing.

Phenolphthalein Eruption ("Verge noir"). Presented by DR. FRED WISE.

V. M., an Italian man aged 37, registered at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital today. He first noticed an eruption on the penis and scrotum two years ago. He has taken proprietary tablets containing phenolphthalein at long intervals during the past two years. Although the lesions would flare up after taking the remedy, he did not suspect the relationship to be one of cause and effect. Yesterday he took a tablet of the drug and felt a burning and itching of the genitalia within a few minutes after ingestion. Within one hour the skin of the penis and scrotum became red and swollen and the itching increased.

He has an oval lesion of the shaft of the penis, about 2 inches (5 cm.) in length and an inch (2.5 cm.) in width, forming a plaque with a dark brown (and in places almost black) border a half-inch (1.3 cm.) in width. The interior of the lesion is bright red and nonscaly. On the scrotum, the skin shows similar sepia-colored and erythematous lesions. There are also faint erythematous plaques on the eyelids and on the lower lip.

MINNESOTA DERMATOLOGICAL SOCIETY

E. M. RUSTEN, M.D., *President*

Minneapolis, April 14, 1939

F. W. LYNCH, M.D., *Secretary*

Pityriasis Lichenoides et Varioliformis Acuta (Haberman). Presented by DR. S. E. SWEITZER, Minneapolis.

L. N., a white woman aged 25, first noticed an eruption around her ankles in September 1938. The eruption occurred in the form of "pimples" and itched severely. She tried many medicines, but with no relief, and the eruption has gradually spread over the entire body. Heat seemed to aggravate her symptoms. Her past health had been excellent.

There are now red and purplish papules densely set over the legs and scattered over the entire body. Many of these have been excoriated, leaving crusts and scars.

(Histologic sections were shown.)

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: I do not want to be a skeptic, but I can hardly accept the diagnosis as presented. The lesions are much more numerous on the legs and appear to me to be follicular pustules rather than papules with central necrosis. I have seen many cases of pityriasis lichenoides acuta et varioliformis acuta, and in all of them the pustules with central necrosis were the characteristic lesions to me, especially when interspersed with scaling papules.

DR. L. H. WINER, Minneapolis: The histologic changes are not those of pityriasis lichenoides et varioliformis acuta, of dermatitis herpetiformis or of urticaria. The epidermis in the involved areas is undergoing changes of hyperkeratinization. There is no vesiculation. The results of histologic examination are inconclusive for a definite diagnosis.

Keratosis Follicularis. Presented by DR. S. E. SWEITZER, Minneapolis.

J. S., a white man aged 66, has had an eruption in the axillas, in the groin and on the scalp as long as he can remember. There have been periods of remissions occurring both spontaneously and as a result of treatment. He has seen many physicians, and many remedies have been tried. He has always been obese, and his general health has been good.

There is a moist papular eruption in the groin and on the genitals, and in the axillas are pigmented areas where the eruption has cleared. The affected areas give off a foul odor. Some areas are covered with a brown greasy crust, and others are red or purplish. The eruption is present in a milder form on areas of the scalp and temple, but there is no tendency to loss of hair.

(Histologic sections were shown.)

The result of a Mantoux test was negative. Examination of the blood revealed hemoglobin, 94 per cent; red cell count, 4,600,000 per cubic millimeter, and white cell count, 19,100 per cubic millimeter; with polymorphonuclear neutrophils, 82 per cent; lymphocytes, 12 per cent; monocytes, 4 per cent, and eosinophils, 2 per cent. Urinalysis gave negative results.

DISCUSSION

DR. L. H. WINER, Minneapolis: Histologic examination shows acanthotic disruptive changes of the lowermost basal cells of the epidermis, with formation of large spaces in the epidermis. There are no *corps ronds*, but after looking at many sections I found one grain cell. This is in accord with views expressed by Pels and Goodman (ARCH. DERMAT. & SYPH. **39**: 438 [March] 1939).

Erythema Nodosum. Presented by DR. S. E. SWEITZER, Minneapolis.

L. M., a white woman aged 32, first noticed three slightly tender red areas over her left shin five days ago. During the next few days new lesions became scattered over the lower parts of both legs, all of which were extremely tender.

For several weeks previous to the onset of the eruption she had not felt well, and for several days the skin on her legs had been burning. Three days before the onset of the eruption her throat became sore, and she suffered from an infection of the upper respiratory tract. She had had a similar attack three years ago.

Examination revealed red slightly elevated inflamed lesions scattered over the anterior surface of both legs. They are hot and tender on palpation. A biopsy was not made.

A roentgenogram of the chest was normal. A Mantoux test and examinations of the blood and urine gave negative results.

DISCUSSION

DR. L. H. WINER, Minneapolis: All patients under my observation with erythema nodosum have had roentgen examinations of their chests and Mantoux tests for the past eight years. No evidence of tuberculosis has been found.

DR. J. F. MADDEN, St. Paul: Several student nurses at the Ancker Hospital have had erythema nodosum. These girls had had negative results of Mantoux tests and of roentgen and physical examinations of the lungs when they started training. In some cases the condition was accompanied or followed by active pulmonary tuberculosis.

DR. H. E. MICHELSON, Minneapolis: In order to evaluate Dr. Winer's remarks about erythema nodosum, one would have to make a careful analysis of his cases. There is no question that there are cases of erythema nodosum in children who have tuberculosis and in children living in families where there is tuberculosis, and in both these groups often an active lesion of tuberculosis cannot be demonstrated, even though the Mantoux reaction is strongly positive. This is certainly an unsettled question, and one must not decide arbitrarily but must remain open minded.

DR. CARL W. LAYMON, Minneapolis: In May 1938 the Dermatologic Reunion of Strasbourg devoted an entire meeting to erythema nodosum. Pautrier championed the view of most dermatologists that erythema nodosum is a syndrome due to diverse causes, while Debré advanced the view of pediatricians and internists that tuberculosis is the predominant and perhaps the sole etiologic factor. Debré's studies of infants with erythema nodosum showed a great deal of evidence for the theory of a tuberculous causation, such as positive results of cultures of the blood and of roentgenologic examinations and the results of tuberculin tests turning from negative to positive after erythema nodosum. Pautrier reconciled these findings with the dermatologists' point of view by admitting that in infants and young children erythema nodosum is practically always tuberculous in origin, while the disease in adults is due not only to the tubercle bacillus but to other infectious agents and toxins. Dermatologists are given much more of an opportunity to observe causes other than tuberculosis, since their patients are not limited to infants and children.

Knuckle Pads. Presented by DR. L. H. WINER, Minneapolis.

M. N., aged 32, noted that the skin on the knuckle of the left index finger became swollen, red and prominent about January 1937. The same condition was noted gradually to affect the left middle and little finger of the same hand, as well as all the fingers of the right hand. The thumbs and toes were never involved.

The skin on the knuckles of the proximal interphalangeal joints of the seven involved fingers shows thickened pads, which are prominent both on extension and

on flexion of the fingers. The condition has neither progressed nor regressed since the patient was first examined, on May 12, 1937.

Roentgenograms revealed no bony changes. Soft tissue tumors of the skin are noted over the involved joints.

Histologic sections showed an increase in fibrous connective tissue in the deep part of the cutis. The elastic tissue was normal in the upper part of the cutis but absent in the areas where connective tissue was increased.

Therapy consisted of occlusive dressing applied for six months, which caused no changes, and roentgen therapy, consisting of 480 r filtered through a 1 mm. aluminum filter in four divided doses from March 21 to April 11, 1939. As yet no changes are evident.

DISCUSSION

DR. L. H. WINER, Minneapolis: I first saw this girl in 1937, at which time Drs. Michelson and Sweitzer also saw her. A biopsy was made at that time and showed definite well circumscribed fibrosis in the deep part of the cutis, almost a fibroma. The condition is an entity closely related to fibroma of unknown cause and frequently a precursor of an associated Dupuytren contraction.

Contraction of the Foreskin. Presented by DR. S. E. SWEITZER, Minneapolis.

J. C., a man aged 72, first noticed tightening of the foreskin about five years ago. This did not cause him any discomfort aside from continual itching. The condition progressed for about a year and one-half, until the external opening became very small. He then consulted a physician. He was circumcised at that time, but about three weeks after the operation he noticed that the skin was becoming indurated again, and the condition has progressed until the present time. His past health has been excellent.

The anterior edge of the foreskin is fixed to the glans near the external meatus, almost encircling it. The skin in the involved area has a whitish sclerotic appearance and is hard on palpation. This has been growing progressively worse, until now the opening is very small and no part of the glans can be visualized.

A biopsy was not made.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: I think the condition is balanitis xerotica obliterans, and in my experience I have been impressed by the contracture of this sclerosing process. In that respect it is different from morphea in other parts of the body and makes one hesitate in alining the two conditions as one. I think that frequent dilation is necessary in this case; it has been my experience that after a period the meatus will remain patent.

DR. S. E. SWEITZER, Minneapolis: The patient had an ordinary circumcision some years ago. The scar became fibrotic. I recommend another circumcision.

Sarcoid of Cheek. Presented by DR. S. E. SWEITZER, Minneapolis.

H. B., aged 34, noted a red spot on the left cheek five years ago, which in the past three years has become hard and has grown. This has been treated for three years with incision, freezing and high voltage roentgen irradiation by another physician.

Examination shows a hard, firm, reddish, discolored nodule in the left cheek, which is adherent to the epidermis and painless.

Biopsy of the nodule showed an area of infiltration of epithelial cells.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: I was glad to see this patient, for I had examined the microscopic section before I saw him. I grant that the histologic picture is suggestive of sarcoid, but there is just enough variation from the classic

picture to make one hesitate in making that diagnosis. I am inclined to think that sarcoid is being diagnosed too often from sections when the pathologic changes do not entirely conform to those accepted for sarcoid.

DR. S. E. SWEITZER: There was a history of five years' duration. On first examination the lesion looked like scleroderma. It had been previously opened twice, the physician thinking that it fluctuated. He has also had some roentgen treatment. The lesion has grown very slowly. Histologic examination showed lobulated bundles of epithelioid cells, and a diagnosis of sarcoid was made.

DR. L. H. WINER, Minneapolis: Clinically, the lesion has the appearance of scleroderma. I agree with Dr. Michelson that the microscopic diagnosis is sarcoid.

News and Comment

AMERICAN ACADEMY OF DERMATOLOGY AND SYPHILOLOGY

The second annual meeting of the American Academy of Dermatology and Syphilology was held on Nov. 6 to 8, 1939, in Philadelphia at the Hotel Bellevue-Stratford. It was attended by 430 members and was the largest gathering of dermatologists which has ever been held in this country. The scientific session opened with an address by Dr. Cornelius P. Rhoads, New York, on "Vitamin B Complex."

Clinical presentations of different diseases were given simultaneously in twenty-one different rooms at the Jefferson Medical College on the second day from 8:30 a. m. to 12:30 p. m. Each presentation lasted one hour, was given four times and was limited to groups of members who had previously indicated their choice of subject. The diseases discussed and the presenters were as follows:

"Tuberculosis, Tuberculids and Sarcoids," Dr. Henry E. Michelson, Minneapolis; "Diseases of Lipoid Metabolism," Dr. Hamilton Montgomery, Rochester, Minn.; "Epithelioma, Nevi and Melanoma," Dr. Eugene F. Traub, New York; "Pigmentary Dermatoses," Dr. Samuel W. Becker, Chicago; "Eczema (All Forms)," Dr. John G. Downing, Boston; "Eczema (All Forms)," Dr. Marion B. Sulzberger, New York; "Acneform Eruptions," Dr. Edward R. Maloney, New York; "Lymphoblastoma," Dr. Harther L. Keim, Detroit; "Lymphogranuloma Venereum and Granuloma Venereum," Dr. Harold N. Cole, Cleveland; "Mycotic Infections of the Hands and Feet," Dr. James H. Mitchell, Chicago; "Mycotic Infections of the Scalp," Dr. George M. Lewis, New York; "Pemphigus and Other Bullous Diseases," Dr. Oliver S. Ormsby, Chicago; "Reactions to Arsenic and Other Bullous Compounds," Dr. Carroll S. Wright, Philadelphia; "Early Syphilis," Dr. Henry B. Decker, Camden, N. J.; "Late Cutaneous Syphilis and Eye Syphilis," Dr. Joseph V. Klauder, Philadelphia; "Syphilis of the Viscera and Bones," Drs. Udo J. Wile, Ann Arbor, Mich., and Charles C. Dennie, Kansas City, Mo.; "Neurosyphilis," Dr. Paul A. O'Leary, Rochester, Minn.; "Congenital Syphilis," Dr. Donald M. Pillsbury, Philadelphia; "Rare Dermatoses," Dr. John B. Ludy, Philadelphia; "Congenital Diseases of the Skin," Dr. Thomas Butterworth, Reading, Pa., and "Mucous Membrane Lesions," Dr. Clark W. Finnerud, Chicago.

Round table discussions of the diseases included in the clinical presentations were held at luncheons on each of the three days of the meeting.

Lectures on special subjects were given in the afternoons by Drs. Joseph V. Klauder, Philadelphia; Loren W. Shaffer, Detroit; Samuel W. Becker, Chicago; Hiram E. Miller, San Francisco; Howard Fox, New York; Jacob H. Swartz, Boston; Fred Wise, New York, and Francis W. Lynch, St. Paul.

Four symposiums were held on the morning of the third day. That on syphilis was in charge of Dr. Udo J. Wile, Ann Arbor, Mich.; allergy, Dr. Samuel M. Peck, New York; pharmaceutic therapeutics, Dr. Otto H. Foerster, Milwaukee, and physiology and chemistry of the skin, Dr. Donald M. Pillsbury, Philadelphia. In addition to those in charge of the symposiums, the speakers included: Drs. James Snow, Ann Arbor, Mich.; Charles R. Rein, New York; Dudley C. Smith,

Charlottesville, Va.; Adolph Rostenberg Jr., Washington, D. C.; J. Gardner Hopkins, New York; Marion B. Sulzberger, New York; Louis Schwartz, Washington, D. C.; Samuel W. Becker, Chicago; Francis E. Senear, Chicago; William H. Guy, Pittsburgh; George C. Andrews, New York; Richard S. Weiss, St. Louis; Elmer R. Gross, Wilmington, Del.; Michael H. Ebert, Chicago; Edward Lodholz, Philadelphia, and Herman Sharlit, New York.

An important educational feature consisted of special courses, which will be continued indefinitely, for at least five years, and others will be added if there is sufficient demand for them. The courses which were given this year for three hours each afternoon and the members who conducted them were: histopathology, Dr. Frederick D. Weidman, Philadelphia; mycology, Dr. J. Gardner Hopkins, New York, roentgenotherapy and radium therapy, Dr. George C. Andrews, New York. Other physicians and technicians assisted in the presentation of the courses.

Dr. Harry R. Foerster, Milwaukee, was elected president, and Dr. Clark W. Finnerud, Chicago, vice president, for the ensuing year. Dr. Earl D. Osborne, Buffalo, and Dr. Clyde L. Cummer, Cleveland, continued to serve as secretary and as treasurer, respectively, having been elected in 1938 for terms of five years. The following members were elected directors to serve for three years: Drs. John G. Downing, Boston; Harry R. Foerster, Milwaukee; J. Gardner Hopkins, New York; Hiram E. Miller, San Francisco, and Richard S. Weiss, St. Louis.

The entertainment consisted of a smoker and a banquet given by the academy. A special feature was a commercial exhibit of special interest to dermatologists, which was presented by eighteen firms.

The next meeting will be held in Chicago in November 1940, the exact date to be announced later.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY

An examination was held in Philadelphia, in November 1939, at the time of the meeting of the American Academy of Dermatology and Syphilology. Seventy-seven candidates passed the examination.

The new diplomates of the board are as follows: Phillip K. Allen, San Diego, Calif.; Joseph C. Amersbach, New York; Leonard A. Anderson, Springfield, Mass.; Leslie P. Barker, New York; Else Anna Barthel, New York; Frederic T. Becker, Duluth, Minn.; George W. Binkley, Cleveland; Eva T. Brodkin, Newark, N. J.; Bertram F. Bruenner, Seattle; Milton G. Butler, Saginaw, Mich.; Thomas Butterworth, Reading, Pa.; Jasper L. Calloway, Durham, N. C.; James F. Campbell, Fort Worth, Texas; C. T. Chiaramonte, Brooklyn; William Harris Connor, Houston, Texas; Frank E. Cormia, Montreal, Canada; G. Marshall Crawford, Boston; David H. Davidson, Brooklyn; Julius D. Davis, New York; Maurice Dorne, Chicago; Everett V. Dulin, East Orange, N. J.; James H. Dwinelle, Rye, N. Y.; Morris M. Estrin, Brooklyn; Isaac Myron Felsher, Chicago; Andrew George Franks, New York; Reuben Friedman, Philadelphia; Walter T. Garfield, Boston; Serafino Genovese, Ridgefield, Conn.; Hyman Joseph Goldman, St. Louis; Charles K. Good, New York; Thomas N. Graham, New York; Irving Greenwald, New Rochelle, N. Y.; Leon H. Griggs, Syracuse, N. Y.; Paul Gross, New York; Arthur F. Hall Jr., Santa Monica, Calif.; Eugene A.

Hand, Saginaw, Mich.; Joseph C. Hathaway, Spokane, Wash.; Egbert J. Henschel, New York; James K. Howles, New Orleans; Harry Keil, New York; Phyllis S. Kerr, White Plains, N. Y.; John H. Lamb, Oklahoma City; Elizabeth Ann Laszlo, New York; William Leifer, New York; Samuel E. Light, Tacoma, Wash.; Eufemio N. B. Lopez, New York; Leslie K. MacClatchie, Washington, D. C.; Morris Markowitz, Philadelphia; Robert R. M. McLaughlin, New York; Charles W. McNitt, New York; Dushan V. Medigovich, Phoenix, Ariz.; James L. Miller, New York; Joseph L. Morse, New York; Emanuel Muskatblit, New York; Hiram D. Newton, San Diego, Calif.; Maurice H. Noun, Des Moines, Iowa; Robert B. Palmer, Lincoln, Neb.; Osgoode S. Philpott, Denver; Morris Polsky, Kansas City, Kan.; Louis L. Praver, Columbus, Ohio; James R. Rogin, Detroit; Dominick L. Romano, New York; Adolph Rostenberg Jr., Washington, D. C.; Heinrich Sarason, New York; Thomas S. Saunders Jr., Portland, Ore.; Julius R. Scholtz, Los Angeles; Harold Shellow, Chicago; Mabel G. Silverberg, New York; Moses Silverman, Brooklyn; Herbert J. Smith, Philadelphia; Maurice Sullivan, Baltimore; Albert S. Tenney, East Orange, N. J.; Norman Tobias, St. Louis; Harold L. Walker, Elmira, N. Y.; George S. Williamson, Ottawa, Ontario, Canada; Maxwell J. Wolff, Los Angeles; Tryphosa R. Worcester, New York.

DIRECTORY OF MEDICAL SPECIALISTS

The first edition of the *Directory of Medical Specialists,* which will list approximately 14,000 specialists who have now been certified by the twelve American boards and the two affiliate boards in the specialties, will be issued by the Advisory Board for Medical Specialties in December. It will be the official publication of the American Board for Medical Specialties and will contain, without expense to them, only the names of those specialists who have been certified by the American boards. It is expected that the directory will be issued every two years.

The directory will contain three sections: (1) a brief description of the Advisory Board for Medical Specialties, its organization and its objectives; (2) a description of the individual boards, with a geographic and a detailed biographic listing of their diplomates, the requirements for admission to their examinations for certification, the details of their organization and other general information, and (3) a complete alphabetic list of all 14,000 diplomates, with their addresses and indications of certification in their specialties.

The editorial board consists of the secretaries of the fourteen American boards. Dr. Paul Titus, 1015 Highland Building, Pittsburgh, secretary of the Advisory Board for Medical Specialists, is the directing editor.

The project is not designed to be profit making; hence the subscription price has been set at $3.50 a copy, which is computed to cover only publication expenses. Subscriptions may be sent to the publication office of the Columbia University Press, 2960 Broadway, New York, or to the office of the directing editor.

PROGRAM NOTICE

Applicants for a place on the program of the Section on Dermatology and Syphilology at the New York meeting of the American Medical Association, June

10 to 14, 1940, are requested to submit titles and provisional abstracts of their proposed papers to the Secretary of the Section, Dr. C. F. Lehmann, 714 Medical Arts Building, San Antonio, Texas.

Applicants need not be members of the Section but must be Fellows of the Association, graduates in medicine not in private practice or invited guests.

TENTH INTERNATIONAL CONGRESS OF DERMATOLOGY AND SYPHILOLOGY POSTPONED

In view of the continuance of the war in Europe, the Tenth International Congress of Dermatology and Syphilology, which was to have been held in New York for a week beginning Sept. 8, 1940, has been postponed indefinitely.

Book Reviews

Recipes and Menus for Allergics. By Myra May Haas, in collaboration with Nathan Schaffer, M.D. Price, $2.50. Cloth. Pp. 286, with illustrations. New York: Dodd, Mead and Company, Inc., 1939.

The subject matter of this book is attractively and simply presented. It consists mainly of a large number of recipes and menus for allergic persons, the purpose of which is to eliminate the offending foods while maintaining appetizing and nutritive diets. It is recommended more for the patient than for the physician and is a handy, ready reference and guide. The task of preparing meals for the allergic patient's family without too great inconvenience is sensibly handled.

A 30 page preface deals with general considerations in regard to the allergic patient, his environment and the offending allergens. The ingredients found in various cereals, farm products, spices, condiments and alcoholic and nonalcoholic beverages are listed.

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